DEPARTMENT OF WILDLIFE

Washington

August 1992

FINAL — SUPPLEMENTAL ENVIRONMENTAL IMPACT STATEMENT LAKE & STREAM REHABILITATIONS —1992-1993

Report #92-14

HABITAT & FISHERIES MANAGEMENT DIVISIONS

FINAL
Supplemental Environmental Impact Statement
LAKE AND STREAM REHABILITATIONS

Description:

The Washington Department of Wildlife (WDW) proposes to continue **Lake and Stream Rehabilitations** to improve fishing for game fish in selected waters via the elimination of other non-game or competitor species of fish.
The following alternative methods area considered in this EIS:
Use of the pesticide Rotenone
Use of other fish toxicants
Use of predator or competitor species
Stocking with legal size fish
Mechanical methods
No Action

Proponent:

Washington Department of Wildlife

Proposed Date of Implementation:

Fall 1992

Lead Agency:

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Licenses Required:

Water Quality Modification - Washington Department of Ecology (DOE)
National Pollution Discharge Elimination System Permit - (DOE)
Approval by Washington Wildlife Commission

Authors and Principal Contributors:

This document was prepared by the regulatory services program, Habitat Management Division, and various staff of Fisheries Management Division, Washington Department of Wildlife.

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Public Hearings:

August 15, 1992

Wildlife Commission
Red Lion Inn
1225 N. Wenatchee Ave
Wenatchee, Washington 98801

Date Final Action is Planned:

September 30, 1992

Type and Timing of Subsequent Environmental Review:

None

Location of EIS Background Data:

SEPA Public Information Center
Washington Department of Wildlife
Habitat Management Division
600 North Capitol Way
Olympia, Washington 98504

Cost to the Public:

None

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SUMMARY

The Washington Department of Wildlife manages approximately 5.9% of the state's lowland lakes throughout the state according to public desires, recreational demands habitat considerations and previous management efforts. Although surveys have shown that trout are the most popular of the state's game fish, some lakes are managed to improve populations of bass, bluegill or crappie. In response to these needs WDW proposes the elimination of non-game or competitor species in a portion of these lakes to allow stocking and optimal populations of trout fingerlings and selected warmwater species. The overall objective of the program is to improve public fishing opportunities.

Alternative Methods:

In Table A the alternative methods are broken into groups; Fish Toxicants, Predator/Competitor, Mechanical and No Action. The methods included in these groups are explained below.

Fish Toxicants:

Rotenone . Rotenone is widely regarded as the safest and least persistent of the poisons.

Other poisons . The list of poisons used to kill undesired fish in lakes and ponds is huge (Lennon et al., 1970; Eschmeyer, 1975). Currently only the Streptomyces derived antibiotic antimycin (marketed as Fintrol) is registered for use as a general fish toxicant with the EPA (Cumming, 1975).

Baits . Baits have been used with limited success, either as an attractant to draw fish to a secluded area of the lake to be eliminated by other means or as a coating for calcium carbide pellets that would produce acetylene gas and float the fish after the bait had been digested.

Predator/Competitor

Predator stocking . Actual experiments with predator stocking as a fish-control technique are scarce, and success has been limited (Dunst, et al., 1974). Both northern pike and largemouth bass failed to control bluegills in Michigan (Shapiro, et al., 1975) and in California (State of California, 1983). California's efforts to control carp, suckers, and squawfish with predators have failed although striped bass reduced shad and bluegills in some reservoirs (State of California, 1983). Since large apex predators would also eat trout fry, this is not an option in most Washington state waters.

Mechanical

Water level drawdown . Completely draining a pond or reservoir is the most foolproof way to destroy all the fish in it (Prevost, 1960); where pockets of water remain, they can be easily and thoroughly poisoned, netted or electroshocked (Barry, 1967). Partial drawdowns that expose carp spawning beds have also been reasonably successful (Sprague, 1961). In Washington State, however, few of the program lakes have water level control facilities.

Lakewide netting and trapping . There are no published accounts of lakewide netting programs that have been successful. Most lakewide attempts using commercial fishermen have failed because they are not cost-effective and are extremely labor intensive.

Dams and barriers . Barriers are used to block migrating fish from their spawning streams. This method has little practical value in Washington where the important target species (carp, perch, and sunfish) are lake spawners.

Electrofishing . Electrofishing on a lakewide basis has never been successful as a control measure and, like netting, is very labor intensive.

Removing congregations of spawning fish . There are several accounts of success with this method, whereby adult fish are allowed to congregate in spawning areas which are subsequently blocked off in most cases. The fish are then poisoned, electroshocked or netted. To actually eliminate a nuisance fish population, this technique would have to be repeated yearly, at least until all year-classes had reached spawning size.

Table A - Comparative Impacts Matrix, by element of the environment.

Method	Significant Impacts	Mitigation Measures
Earth		
Fish Toxicants Predator/Competitor	Rate and distribution of lake soil sediment may be altered with changes in species abundance and diversity	
Mechanical	Change in sediment transport through/around dams or barriers Changes in plant/benthic from drawdown	
Air		
Fish Toxicants Mechanical	Adverse odors may be present while fish killed decompose	Extended fishing season to increase opportunity to harvest fish prior to rehabilitation
Surface Water		
Fish Toxicants Mechanical Predator/Competitor	Changes in bacteria levels and turbidity, change or elimination of phyto/zoo-plankton, benthic fauna, fish species and diversity, algae blooms, change to water taste and odor. Algae blooms.	Lakes can recover from algae blooms, loss of phyto/zoo-plankton, benthic fauna and changes to taste and odor in two to twelve months.

Table A. continued.

Method	Significant Impacts	Mitigation Measures
		Mitigation measures include actions to restrict the use of rotenone to targeted waters only, and to include potassium permanganate dip stations and temporary sand bag dams Lakes would be restocked with desired species.
Terrestrial Resources		
Fish Toxicants Predator/Competitor Mechanical	Larval amphibians and some adults may be killed. Adult amphibians or reptiles may be temporarily affected by loss of aquatic fish food source. Birds or mammals which depend on fish/benthic organisms for food may be temporarily impacted	Treatments are timed to produce the desired rehabilitation with the least impact to other species
	Humans in direct contact with the powder rotenone may experience temporary skin, eye or mucous membrane irritation.	Protective clothing
Environmental health		
Fish toxicants Predator/Competitor Mechanical	The rotenone label precludes the consumption of rotenoned fish as food or feed.	Disposal of fish or prevention of use.

Table A. continued.

Method	Significant Impacts	Mitigation Measures
	Increases in human activities as a result of increased fishing pressure may cause erosion, air, water and noise pollution, trampling of vegetation, and other impacts to recreational, religious, or scientific use of the area.	Monitoring, education and enforcement.
Aesthetics		
Fish toxicants Mechanical	Water will be brown in color following treatment with rotenone or disturbance by mechanical means.	Water will recover in a few hours to a few days.
	Floating or beached fish	Disposal or education.

DESCRIPTION OF THE PROPOSED ACTION:

Type of Action

The proposed action is to continue the Washington Department of Wildlife's rehabilitation of selected lakes and streams by eliminating undesired fish species using rotenone followed by restocking with a preferred fish species, to improve public fishing. Lake and stream rehabilitations occur throughout the state. Almost all treatments have occurred in lakes and ponds, with only occasional stream or slough treatments.

In the last 20 years approximately 5.9% of the state's lowland lakes have been treated with rotenone. This equals about 3.4% of the state's total standing water acreage below 2500 feet in elevation. The average per year has been 0.3% of total surface acreage of the state's lowland lakes.

Justification

Fisheries Management

To satisfy the annual demand for productive gamefish fishing by over 600,000 anglers, Washington Department of Wildlife stocks selected waters with trout from hatcheries and transplanted bass, crappie, walleye, and additional warmwater fish species from other waters. Many waters are managed for specific fisheries, such as trout only or warmwater species. The management emphasis for state waters is decided according to habitat parameters, public desires, recreational demands, and previous management efforts. Occasionally, these waters become overpopulated with fish species outside this management emphasis. This often results in increased predation and/or competition, hence poor growth and survival, of targeted game fish. If carp overpopulate, fish survival decreases and nesting bird habitat is degraded due to siltation and uprooting of emergent vegetation. Infestations of these fish species occur through migration from other waters or through illegal transport and introductions. Three management options are available if this happens:

1) Take no action;
2) Change the management emphasis for the water;
3) Eliminate competing species and stock with desired gamefish species.

Option 1 will result in an increase in numbers of fish outside the management emphasis to a point where the water no longer supports a viable gamefish fishery.

Option 2 allows for a viable fishery, but is relatively costly. For example, to establish a trout fishery, the cost of producing a fingerling trout in a state hatchery is about 4% of the cost of a legal-sized trout (Washington Department of Wildlife, 1983). Even though fry survival is lower when compared to legals, they can still be more economical in some cases (see below). Furthermore, legal-sized trout are considered a lower quality fish than naturally-reared fry-origin trout, and are usually smaller as well.

Option 3 is the only alternative that allows the lake to continue to provide a viable fishery. Rotenone is the method currently used by WDW to eliminate fish in lakes and is far more economical than either options 1 or 2 above. Washington Department of Wildlife (1984) compared the costs of three different management strategies for a typical lowland trout lake in western Washington (Lake Erie, Skagit County).

These options were:

1) trout-only lake maintained by fry stocking and periodic rotenone treatment;
2) mixed-species lake maintained by trout fry stocking (no rotenone); and
3) mixed species lake maintained by legal-sized trout stocking (no rotenone).

The cost of Option 1 was about one-third the cost of either Option 2 or 3. Also note that Option 2 is not likely to be a viable alternative in many lakes for the reasons already discussed.

Wildlife Management

Lakes are also rehabilitated by the Department of Wildlife to improve the quality of waterfowl habitat. The primary objective is to remove carp from potentially productive nesting and rearing duck habitat to increase the amount of food (aquatic invertebrates) and vegetative cover. Candidate waters are primarily one to three feet deep.

Pre-Treatment Procedures

A lake or stream is selected for rotenone treatment when a viable fishery can only be provided with introductions of legal-sized fish. These determinations are made by the WDW Area Fisheries Biologist directly charged with managing the lake's gamefish. Standard indicators of fishery performance are the average catch per hour on opening day, and fish size and abundance from annual pre-season gillnet sets. When poor performance is coupled with gillnet and/or electroshocking data showing and increase in species

outside the managment emphasis, the Area Biologist may recommend treatment to his Regional Biologist.

A Pre-Rehabilitation Plan (See Appendix A) containing vital information on the proposed treatment must be completed by the biologist.

In calculating the dosage of rotenone needed, the biologist considers a variety of physical and biological factors, the most important being target species, water chemistry, past successes or failures in the lake and presence of weedy shorelines.

Dosage is initially calculated based on powder or liquid containing 5% rotenone, and is expressed as parts of powder or liquid - not pure rotenone itself - per million parts of lake water (ppm) on a weighted basis. One ppm is equivalent to one milligram per liter (1 mg/l).

The powders used by WDW rarely contain only 5% rotenone. WDW receives most of its rotenone dust from Peruvian suppliers, and shipments are chemically assayed by batch for rotenone content. Powders used from 1977 through 1984 ranged from 6.6% rotenone to 8.1% rotenone. Liquid preparations consistently contain 5% rotenone. When these formulations are received and the exact assay known, biologists adjust the amount of powder used to conform to the initial calculation based on 5% powders.

The actual amount of rotenone needed is based on the estimated weight of water in the lake. This is determined by volumetric calculations using WDW surveys on the particular lake.

The Regional Fisheries Program Manager presents his list of proposed treatments along with justifications for each water to the Fisheries Management Division of WDW. Approval at this stage may depend not only on the validity of the biological justifications, but on other considerations such as the lake's public use and its importance as a recreational fishery, and the availability of rotenone itself. Statewide priorities are established, and a list of candidate lakes drawn up.

After developing a list of candidate lakes, the public is notified through a general news release, usually in late spring. Area Biologists also solicit public opinion from lakeshore residents and other groups in the area. Public meetings are held in the vicinity of the waters proposed for treatment prior to a final decision.

At its annual August public hearing, the Washington State Wildlife Commission - a group of private citizens chosen by the Governor to oversee WDW - is presented with the list of candidate lakes. The Commission approve or denies treatment on individual lakes at its annual August meeting. Even after a lake has been approved by the Commission, WDW may opt not to treat that lake.

Treatment Procedures

Shortly before treatment, the lake is divided into sections of similar volumes, and these sections are marked using buoys and shoreline markers.

On the day slated for treatment, each section of the lake is assigned to a WDW employee. Rotenone is applied by towing burlap sacks of commercial dust behind a boat, the outboard prop wash helping to diffuse the poison. Shoreline and marshy areas are often sprayed with liquid rotenone by motorized pump or are dusted by hand. Aerial applications are sometimes made. Common dosages of rotenone (5%) in lakes treated in Washington range between 1-4 ppm.

Fishing regulations are liberalized when possible, and upon approval by the Wildlife Commission, to utilize fish in waters scheduled for rehabilitation. Warmwater game fish, usually mature bass, are collected (depending on need) prior to rehabilitation, to be utilized as broodstock for waters nearby which are managed for warmwater fisheries. On some lakes, bass that have floated to the surface have been netted by WDW employees and bass club volunteers, revived by dipping the fish in potassium permanganate, and moved to mixed-species or spiny ray lakes to augment or start a population (Fletcher, 1976). WDW has typically transplanted 200-300 fish from a single lake during this type of procedure. The use of potassium permanganate also requires a short-term water modification (permit) to the water quality standards issued by the Washington Department of Ecology.

Post-Treatment Procedures

In lakes with a stream outlet, runoff from the lake must be controlled or detoxified. In some cases, the runoff is small enough that it can be dammed off (using sandbags, for example) until the rotenone is naturally degraded. When this is not possible, and oxidizing agent - usually potassium permanganate - is dripped into the outlet stream to detoxify the rotenone before it can harm fish and invertebrates downstream. Between 1977 and 1984 such detoxification was necessary in only 16% of the lakes treated. Pfeifer (1985) provides a detailed account of outlet detoxification procedures, including dosage/detoxification curves and case histories in Martha and Silver Lakes (Snohomish County).

In the lake itself, rotenone degrades naturally over time. At intervals following treatment, WDW Area Biologists usually perform a simple bioassay to determine how long the lake remains toxic to fish: hatchery rainbow trout are commonly suspended in the water column in wire cages and when these fish survive 1-6 days in the lake, it is considered nontoxic.

The biologist submits a Post-Rehabilitation Form (see Appendix A) for each treated water; it describes, among other things, the possibility of a complete kill, water conditions at the time of treatment, and any detoxification measures taken.

Fish are restocked the following spring. During the post-treatment years, the Area Biologist continues to monitor fish survival and growth, as well as catch rates for the water.

Number and size of Waters Treated

The fist rotenone treatment in Washington State took place in September, 1940 on King Lake (Pend Oreille County). Since that time 473 state waters have been treated at least once. The chlorinated hydrocarbon insecticide toxaphene was occasionally used instead of rotenone; its use was discontinued in the late 1960's, and since then, rotenone has been the only fish poison applied in Washington State.

Almost all treatments have occurred in lakes and ponds, with only occasional stream or slough treatments. Waters treated since 1940 represent [5.72%] of the total surface acreage of all lakes below 2,500 feet elevation in the state.

Frequency of Rotenone Treatments

Rotenone rarely if ever kills all the fish in a lake. Problem species often repopulate the lake naturally over the course of time. In addition, problem species are often reintroduced illegally by anglers or lakeside residents. These may be the same species that originally degraded the targeted fishery, or new ones. The net result of any of these cases is the same: fish production will eventually decline, and the lake may have to be rehabilitated again.

Of 473 Washington State lakes that have been treated, 240 (55%) have been treated more than once. The average length of time between treatments has been 7.74 years ($n = 522$ intervals, $s = 4.49$ years).

Target Species

In the eastern half of the state pumpkinseed sunfish was most frequently targeted for elimination, in the western half of the state yellow perch was most frequently targeted. Other important target species statewide include carp, crappie, brown bullhead (catfish), and largemouth bass. All are introduced, non-native species.

A particular lake may experience recurring problems with the same target species over the course of many years. Often, however, the target species on frequently-rotenoned lakes changes over the years. This is often the case in "urban" lakes which are frequent targets for illegal fish introductions.

Timing of Rotenone Treatments

Seventy-eight percent of rotenone treatments in the state have taken place in the fall, mostly in September and October. Only 22% have been spring treatments, and these occurred mostly in March. All spring treatments were on eastern Washington lakes.

Rotenone is usually applied in the fall because water levels are low, aquatic vegetation is sparse, recreational use of the lake is reduced and since most lake's summer thermal stratification has ended (allowing rotenone to circulate throughout the water column). Spring rotenone treatment are occasionally performed on certain lakes with extensive shallow or weedy areas; higher water levels in the spring make these areas more accessible by boat.

Legal Standing

RCW 77.12.420 empowers the Wildlife Commission to eradicate "undesirable types of fish. The Commission's right to rehabilitate lakes and streams was affirmed by Thurston-Mason County Superior Court in the case of Patrick vs. Biggs (#27476), January, 1954.

Funding

Lake and stream rehabilitation operations are funded through fishing license fees and through taxes collected by the federal government on fishing tackle at the manufacturing level and apportioned to states under the Dingell-Johnson Act. Dingell-Johnson funds are limited to 755 of total project costs. A 25% contribution on Department of Wildlife monies is required by federal law. Lake and stream rehabilitation with rotenone is an approve fishery management activity under Dingell-Johnson funding.

DETAILED ASSESSMENT OF IMPACTS

Earth

Lake and stream rehabilitation may have some effect on lake soils since changing diversity of fish can influence rate and distribution of organic sedimentation. No specific data are available on this subject.

By enhancing fishing in a lake, more fishermen may visit the area. Increased human activity may also increase erosion if vegetation becomes trampled and undeveloped trails are used more frequently.

Air

Rotenone droplets or mist may be carried in the air from the liquid applications. Powder rotenone is applied by towing an open sack underwater, so escape of particles in the air should be minimal. Decomposing fish emit an adverse odor to the surrounding atmosphere. Since the rate of decomposition is influenced by temperature and moisture, rehabilitation projects are usually scheduled during periods that minimize the undesirable aspects of decomposition. In residential areas, dead fish are sometimes used in gardens and flower beds as fertilizer by local residents.

Also better fishing in an area usually attracts more people during fishing season. This may increase noise and air pollution from cars and boats.

Water

From a human use standpoint, important water quality parameters in lakes include dissolved oxygen, fecal coliform levels, total dissolved gas, temperature, Ph, turbidity, and aesthetic values (Title 173 WAC, Water Quality Standards, pages 187-1988, 1983). Where lakes supply drinking water for people or livestock, safety and palatability of the water are obvious concerns. A variety of other chemical and biological parameters are also considered here as water quality factors.

Some important aspects of water quality that are affected indirectly by rotenone treatment include phytoplankton, which affects water transparency and thus aesthetic values and dissolved oxygen levels at the sediment/water interface, and the effect of fish stocking on lake phosphorous loads.

There has been only one comprehensive study of how rotenone treatment indirectly affects all routinely-measured water quality parameters : Bonn and Holbert (1961) conducted tests on 18 water quality indicators in Lake Lavon and Bonham State Park Lake, Texas. Their goal was to determine the indirect effects of rotenone treatment on municipal drinking water supplies. Only coves in Lake Lavon were treated, with non-treated coves serving as controls. In Bonham State Park Lake, all 49 acres were treated for a complete fish kill and results were compared with pretreatment data. Standard rotenone formulations and dosages were used, and after dead fish were weighed, their carcasses were punctured and scattered back into the water to create a natural post-treatment environment. Samples of water were taken from various depths at two-week intervals during the year, and at shorter intervals immediately prior to and after the treatment. Bonn and Holbert tested the following parameters.

1) temperature
2) Ph
3) turbidity
4) dissolved oxygen
5) carbon dioxide
6) total alkalinity
7) calcium
8) NH_4
9) organic nitrogen
10) total nitrogen
11) phosphorous
12) potassium
13) total phytoplankton
14) generic makeup of phytoplankton
15) total hardness
16) odor number
17) most probable number (of coliform bacteria)
18) bacterial colonies per milliliter

Of these 18 parameters, only four showed significant change due to the treatment: turbidity decreased, phytoplankton increased, noncoliform bacteria increased, and the water took on a disagreeable taste and odor. The change in taste and odor of the water was by far the greatest of the water quality changes noted.

Scattered water quality data from other studies (which gathered them from ancillary information) are also available:

Brown and Ball (1943a) measured water temperature, dissolved oxygen, carbon dioxide, methyl orange alkalinity, and pH throughout the water column in Third Sister Lake, Michigan. None of the factors changed significantly within four days of rotenone treatment when compared to pre-treatment data.

Houf and Campbell (1977) compared three small, fishless Missouri ponds treated with rotenone and two untreated control ponds, concluding that rotenone treatment "had no noticeable effect on water chemistry." The monitored pH, water temperature (pond surface and bottom), dissolved oxygen (pond surface and bottom), hardness and alkalinity. These parameters were measured throughout the experiment, which began three months before treatment and ended a year after treatment.

Wollitz (1962) measured several chemical and physical properties of two Montana ponds before and after rotenone treatment. He found that oxygen saturation, alkalinity, pH, nitrate, and inorganic phosphate levels did not change significantly after treatment. In one of the ponds turbidity decreased and transparency increased after poisoning.

Bandow (1980) found no significant changes in the surface temperature, dissolved oxygen (surface and subsurface), or nitrate nitrogen levels in Carls Lake, Minnesota, after it was poisoned with rotenone. Transparency increased dramatically, however, due to lower algae levels.

Based on these studies and those of Bonn and Holbert (1961), it can probably be concluded that water quality parameters unaffected by rotenone treatment, either directly or indirectly are: water temperature, dissolved oxygen, pH, alkalinity and carbon dioxide.

Those water quality parameters that have been shown to be affected indirectly by rotenone treatment are:

1) Phytoplankton levels - Both increases and decreases in the level of phytoplankton have been documented following rotenone.

2) Bacteria levels - Bonn and Holbert (1961) saw an increase in the number of bacteria per milliliter in both Texas lakes they rotenoned. They felt the increase could be due to the decay of dead fish and/or the agitation of the water and bottom sludge during the treatment. Since there was no corresponding increase in the Most Probable Number of coliforms, bacteria other than coliform constituted the increase. The bacterial increase was temporary, and the authors noted that most modern water treatment plants could cope with it without difficulty.

3) Turbidity/Transparency - Turbidity in water is caused by suspended matter, either organic or inorganic (American Public Health Association, 1971). Strictly speaking, it is not the same thing as transparency or visibility (usually measured by Secchi disc), though it is obviously related. In lakes that are turbid because bottom-scavenging fish constantly stir up sediments, poisoning with rotenone or other toxicants almost always results in reduced turbidity. However, in a deep lake with a coarse or gravelly substrate, turbidity from bottom-scavenging fish is not likely a problem. It is possible that nutrient re-suspension resulting in bloom conditions following a rehabilitation can reduce water transparency, although no studies were found to substantiate this speculation.

   Increased water transparencies following carp poisoning have been reported in lakes in Illinois (Bennett, 1943), North Dakota (Needham, 1966), Colorado, (Tanner and Hayes, 1955),

Ohio (Weier and Starr, 1950), Wisconsin (Klingbeil, 1975), and Oklahoma (Eschmeyer, 1953). In Bass Lake, Indiana, removing the carp by seining produced the same results (Ricker and Gottschalk, 1940). None of these results were quantified and only refer to increased "visibility" making it difficult to determine which if two important factors - suspended silt, or algae - was responsible for the improvement. Other work has shown that carp and other bottom-feeders cloud the water not only by stirring up mud but also by increasing algae levels, and that the latter may be far more important in some lakes (Lamarra, 1975; Smeltzer and Shapiro, 1982). Some researchers specifically mentioned reductions in suspended silt or mud as the reason for improved water clarity (Cushing and Olive, 1957; Hoffman and Olive, 1961; Hoffman and Payette, 1956).

Only two studies have actually quantified turbidity (as distinct from transparency) following rotenone treatment: Bonn and Holbert (1961) recorded an 85% reduction in turbidity five days after poisoning Bonham State Park Lake, Texas. Wollitz (1962) cited a 54% drop in turbidity in Middle Pond, Montana. In both cases, the authors attributed the improvements to the elimination of bottom feeding fish. Wollitz (1962), however, reported no turbidity changes in a nearby pond containing few bottom-feeders that was also poisoned.

While decreased turbidity is generally considered a good thing, Bonn and Holbert (1961) suggested that clear water might allow a surge in algae growth. They cautioned that this would be undesirable in drinking water supplies if the algae consisted either of unpalatable blue-greens, or filter-clogging forms.

4) Water Taste and Odor - Researchers at several municipal water supplies have reported changes in the taste and odor of rotenone-treated water.

Of the 18 water quality tests performed by Bonn and Holbert (1961) on two Texas lakes, the greatest changes occurred in water taste and odor. They rated these changes using the Odor Number Test established by the American Water Works Association (American Public Health Association, 1971). Drinking water normally rated a "5-musty" before treatment changed to a "30-kerosene" odor number the day following treatment with rotenone. This was attributed to the hydrocarbon solvents in the rotenone formulations (Noxfish and Chemfish Special). Five percent rotenone powder produced no such kerosene odor in treated water. The kerosene odor disappeared five days after treatment.

A fishy odor was detected 17 days after treatment in one of the Texas lakes. The odor number in a treated lake cove became as high as "30-fishy" three days after treatment, then

disappeared six days later. These changes obviously occurred as a result of decaying fish.

Since it contains no petroleum-based carriers, Bonn and Holbert (1961) recommended 5% rotenone powder as a first choice when treating drinking water supplies. Their laboratory tests confirmed that rotenone powder by itself produced no change in odor number.

While both the kerosene and fishy odors were temporary, Bonn and Holbert's (1961) lab tests showed that both odors could be eliminated by a 1.0 ppm of activated carbon for each threshold odor number produced.

Cohen et al. (1960; 1961a; 1961b) made detailed laboratory and field tests of rotenone in drinking water supplies. They also concluded that the solvents, rather than rotenone itself, caused the kerosene odor. Like Bonn and Holbert, they concluded that activated carbon was the most effective way to reduce obnoxious odors resulting from emulsified rotenone formulations. Depending on the commercial rotenone formulation used, between 36 and 85 ppm activated carbon would be needed to make water with 2 ppm formulation immediately palatable.

Residual Toxicity in Drinking Water - Municipal drinking water supplies have been treated with fish-killing concentrations of rotenone in at least six states, with no harmful effects: Texas (Bonn and Holbert, 1961); Massachusetts (Stroud, 1956); California (Hoffman and Payette, 1956; State of California, 1983); Oklahoma (Eschmeyer, 1953); Indiana (Barry, 1967); and North Dakota (Cohen et al.1961b). In some cases, rotenone treatment has been used specifically to improve or protect the drinking water quality (Hoffman and Payette, 1956; Barry, 1967). Cohen et al. (1960; 1961a; 1961b) performed the most extensive research on the effects of rotenone in public drinking water, and they concluded that rotenone treatment was "consistent with the objective of a water treatment: namely, to produce a safe and potable water".

Despite rotenone's relative safety, the U.S. Environmental Protection Agency (EPA), as a matter of policy, does not set tolerances for pesticides in drinking water. States such as California therefore require that whenever drinking water reservoirs are treated, that the rotenone be detoxifed to undetectable levels (less than 0.005 ppm pure rotenone; Dawson et al., 1983) before it reaches the public. Detoxification can occur through natural breakdown, chemical treatment or both (State of California, 1985).

Rotenone breaks down quickly in the environment (Schnick, 1974), and retention time is long enough in most public reservoirs to allow complete natural detoxification (Bonn and Holbert, 1961;

Cohen et al., 1960). There are occasions when water may reach the treatment plant with some residual toxicity. Although there is little likelihood that it could have any effects on humans or livestock (Cohen et al., 1960; U.S. EPA, 1981), this residue must be removed, or chemically altered, to produce a finished drinking water of good quality. Cohen et al. (1960) made detailed recommendations for eliminating any residual toxicity using activated carbon. They also tested their laboratory finding in a drinking water supply in North Dakota (Cohen et al., 1961b), using 61 ppm activated carbon to detoxify a water supply treated with 2 ppm rotenone.

Both the State of California (1985) and the National Academy of Science (1983) have computed "safe" levels of rotenone in drinking water. California's figure was in the form of an Action Level (AL = the concentration of material in water above which human health may be adversely affected), while the Academy computed a Suggested No-Adverse-Response Level (SNARL). Both the AL and SNARL were based on long-term dosing study of the Midwest Research Institute (1980). Both California and the Academy applied a safety factor of 1,000 to the study's no-effect levels (10 for variability within species, 10 because the study was less than a lifetime, and 10 because the study is to be applied to humans). The SNARL for a 150-pound person who drinks half a gallon of treated water per day was 0.014 ppm pure rotenone; California's more conservative AL for a 22-pound child who drinks a quart of treated water per day was 0.004 ppm.

The detection of pure rotenone in water is approximately 0.005 ppm, slightly below the SNARL and slightly above the AL. The State of California (1985) therefore concluded that a conservative and justifiable requirement for human safety would be that no measurable levels of rotenone be allowed in public drinking water.

Effects of Trout Stocking - Bottom-feeding fish directly influence turbidity levels, and indirectly influence algae levels. Planktivorous fish - among them both stocked trout and numerous "target species" for rotenone - can also exert an indirect influence on algae. Algae levels, in turn, can affect the levels of ammonia, hydrogen sulfide, and hypolimnetic oxygen in a lake.

A lake stocked with trout or any other planktivorous fish will generally support higher algae levels than the same lake if it were fishless. This may partially offset by the periodic removal of other planktivores (e.g., perch or bluegills) with rotenone, and possibly the removal of nutrients from certain lakes through trout angling.

In annual stocking of trout-only lakes in Washington state, no change beyond those which have historically occurred as part of previous rehabilitation and stocking of trout only lakes in any

water quality parameter would be expected due to a post-rotenone introduction of the same (i.e., historical) magnitude.

Plants

According to most studies, phytoplankton is not directly affected by rotenone at concentrations of up to 3 ppm of the 5% dust (Bandow, 1980; Anderson, 1970; Wrenn, 1965; Kiser et al., 1963; Bonn and Holbert, 1961; Hooper, 1948; Smith, 1940; Smith, 1941; Brown and Ball, 1943a; Stenson, 1972).

Only two authors have reported toxic effects on phytoplankton: Wollitz (1962) stated that *Dinobryson* was absent for two weeks in a Montana pond treated with 0.7 ppm Pro-Noxfish. It returned to its former abundance two weeks later, and no other phytoplankters were affected. Almquist (1959) reported that concentrations of 5% rotenone above 2 ppm killed all *Volvox*, while 1 ppm was capable of destroying *Ceratium*. Anderson (1970), however, noted no decrease in either genus when subjected to 0.75 ppm.

Indirect Effects of Rotenone and Trout Stocking - It is difficult to summarize the indirect effects of rotenone and subsequent trout stocking as there are a greater number of trophic links involved.

Figure 1 is a flow chart showing the most important ways in which rotenone poisoning and subsequent trout introductions may influence lake algae levels. It is assumed for simplicity's sake that the two main factors that influence algae growth are the amount of phosphorous (P) avaialable and the level of grazing by zooplankton.

While productivity in some lakes is limited by other nutrients (e.g. nitrogen, silicon, CO_2) algal growth in most culturally eutrophic lakes is controlled by the amount of phosphorus available (Schindler, 1974; Vollenweider, 1968). Within the limits normally found in lakes, Figure 1 illustrates the valid generalization that when phosphorus increases, so do algae levels; when phosphorus decreases, algae is reduced.

There is also ample evidence in the literature supporting the second assumption made in Figure 1: increased grazing by zooplankton generally crops down algae, while decreased grazing boosts algal biomass (Gliwicz, 1975; Shapiro et al., 1975). There are important exceptions, the first to be discussed is the pathways in which rotenone and trout stocking affect phosphorus levels. Rotenone treatment of a lake potentially affects phosphorus levels in two ways:

1) the numbers of bottom-feeding fish (such as carp and bullhead) decrease, which in turn may reduce phosphorus levels; and

2) dead fish decay on the lake bottom releasing the phosphorus

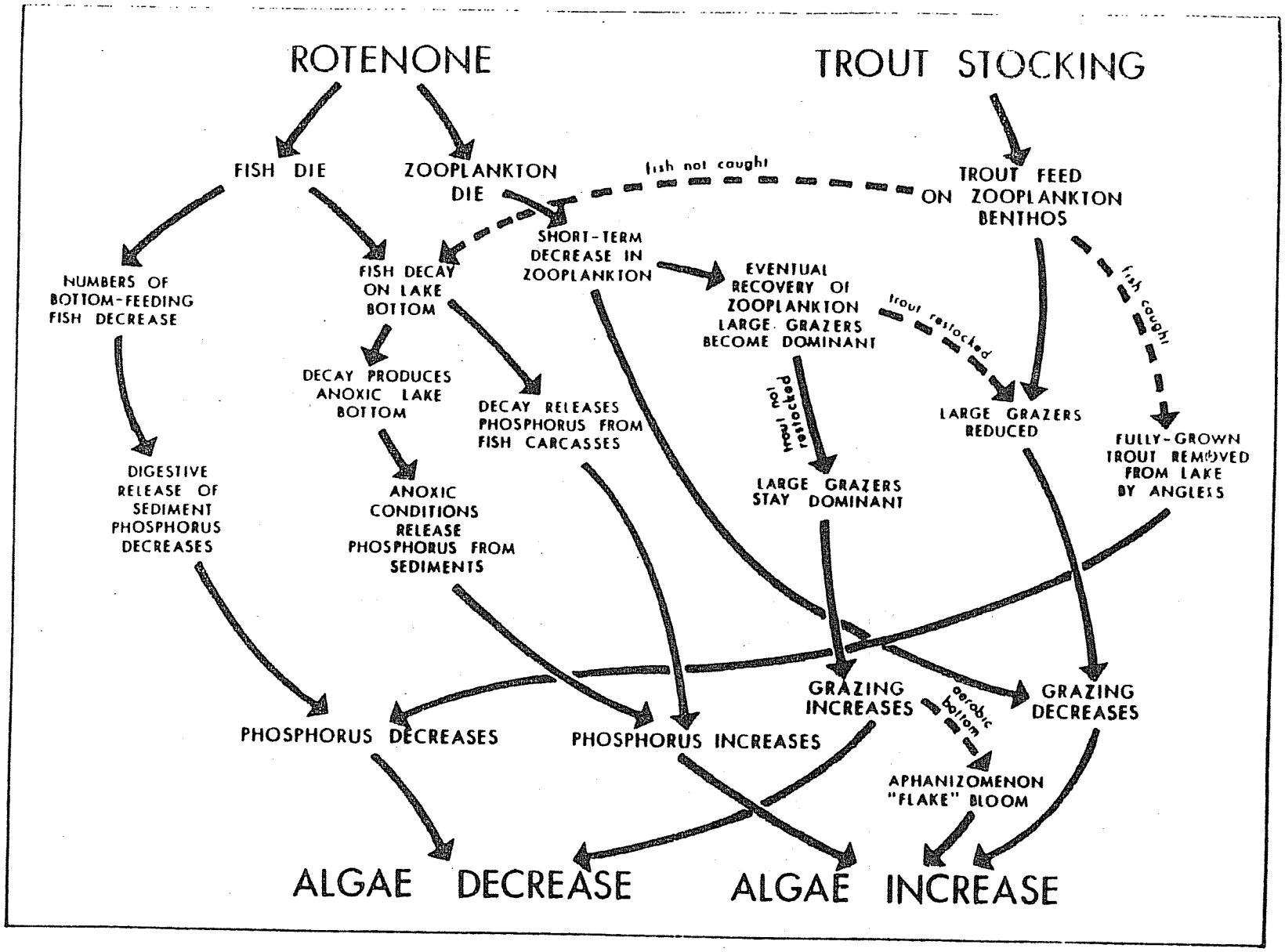

Figure 1. Flow chart showing the most important ways in which rotenone poisoning and subsequent trout stocking may affect algae levels in a lake.

bound in their carcasses and possibly creating anoxic bottom conditions which could release phosphorus from the lake sediments.

Trout stocking also affects phosphorus levels in two ways:

1) trout not caught by anglers die and decay on the bottom, increasing phosphorus as in (2) above; and

2) trout caught by anglers represent a loss of phosphorus from the system.

Effect of Bottom-Feeding Fish on Phosphorus Levels - Bottom-feeding fish such as carp, goldfish, and bullheads have for years been associated with murky water (Moyle, 1968). Some of the reduced transparency is due to suspended silt stirred up by the fish as they scavenge the bottom especially in shallow lakes. But algal blooms associated with these fish can play an important, if not overriding role in clouding the water.

It is possible to separate the effects of silt and algae by plotting the reciprocal of secchi disk transparency against chlorophyl concentrations (Brezonik, 1978); the intercept of the regression represents the amount of murkiness due to substances other than algae (e.g. silt) in the water. Smeltzer and Shapiro (1982) did this in a carp and bullhead infested Minnesota lake, and found that most of the light attenuation (71%) was caused by algae; stirred-up silt was only a minor contributor.

Empirical evidence that bottom-feeding fish can cause algae blooms comes from lakes where these fish have been poisoned: Hoffman and Payette (1956) killed 107 tons of carp with rotenone in a San Diego reservoir and within a month noted marked decreases in most algal counts and increased transparency (though a diatom bloom took place). Needham (1966) found that chlorophyta decreased steadily and remained at low concentrations after posioning bottom fish in North Dakota lakes. Bandow (1980) reported that reduced algal levels followed bullhead removal in a number of Minnesota lakes. Hrba'cek et al. (1961), Stenson et al. (1978), and Schindler and Comita (1972) have all documented similar improvements following the demise of bottom-feeders.

It was once widely accepted that bottom fish release nutrients (such as phosphorus) into the lake by stirring up the bottom sediments; in turn, these nutrients fostered algae blooms. While agitation does release phosphorus (Zicker et al., 1965), there is usually more phosphorus absorbed by aerobic sediments than lost (Fitzgerald, 1970); if bottom fish were releasing phosphorus and causing algal blooms, some other mechanism must be involved. Using carp, Lamarra (1975) proved that it was mostly the digestive activity of bentivorous fish that released phosphorus from the sediments and, more importantly, raised chlorophyll levels. Simple

mechanical stirring of the bottom, on the other hand, did not release appreciable amounts of phosphorus nor did it increase algae levels. Lamarra also showed that release of phosphorus in all its forms was negatively correlated with fish size (i.e., bigger carp release less phosphorus) and that 50% of the total phosphorus excreted by all sizes of carp was in the form of orthophosphate, which is immediately available for algal growth, Figures 2 and 3 display the relationships between carp size, carp density, and sediment phosphorus release from Lamarra's experiments. The actual excretion rate of dissolved phosphorus for a specific weight class of carp or bullhead may be computed from Lamarra's regression equations:

1) for carp: $\log_{10} E(DP) = -.49 \log_{10} W + .027T + .77$

2) for bullhead: $\log_{10} E(DP) = -.379 \log_{10} W + .027T + .344$

where:

E(DP) = specific excretion rate of dissolved phosphorus (micrograms/gram wet weight per hour)
W = wet weight of fish (g)
T = temperature (°C)

With an estimate of fish biomass for various size classes in a lake, it is possible to compute the annual phosphorus loading due to carp and bullhead. Lamarra performed these calculations for the typical "rough-fish" lake in Minnesota. Such a lake contains about 200 kg of carp/ha, and Lamarra estimated that they recycled between 1.07 mg and 2.18 mg total $P/m^2/day$, or 0.52 mg orthphosphate/m^2/day. Even the smaller, more conservative estimate is surprisingly high, and Lamarra concluded that carp were probably liberating amounts of sediment phosphorus that were significant in terms of the lakes' total phosphorus budgets.

In view of its ability to liberate large amounts of phsophorus from lake sediments, Lamarra termed the carp a "phosphorus pump". This ability is not confined to carp alone; the bullhead is also an important "phosphorus pump" (Lamarra, 1976; Shapiro et al., 1975; Bandow, 1980). Although no quantitative data exist, we can probably add the goldfish to this list in view of its genetic similarity to the carp (the two interbreed in the wild).

Smeltzer and Shapiro (1982) further investigated the significance of these experimental findings in a lake dominated by black bullheads and carp. They found that bullheads at a density of 59 kg/ha and carp at 43 kg/ha were contributing 88 mg of P/m^2/year to Lake Marion, Minnesota. This same eutrophic lake was receiving 84 mg of P/m^2/year from external sources. The conclusion that benthivorous fish were supplying the lake with as much phosphorus as all external sources combined (drainage, rain, and septic tank seepage) is astounding, and implicates them as major contributors to algae blooms.

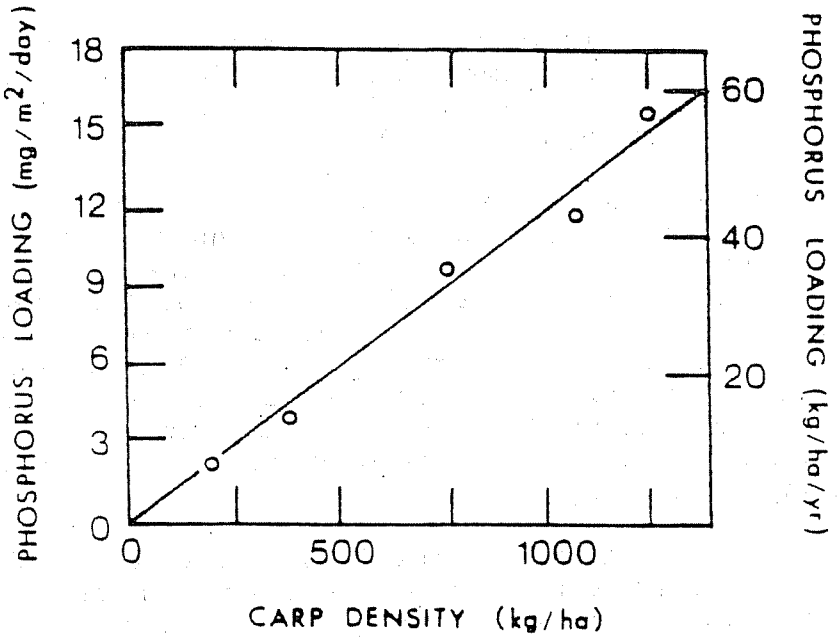

Figure 2. Relationship between carp density and phosphorus release. Carp were between 140 and 180 grams wet weight. Source: Lamarra 1975.

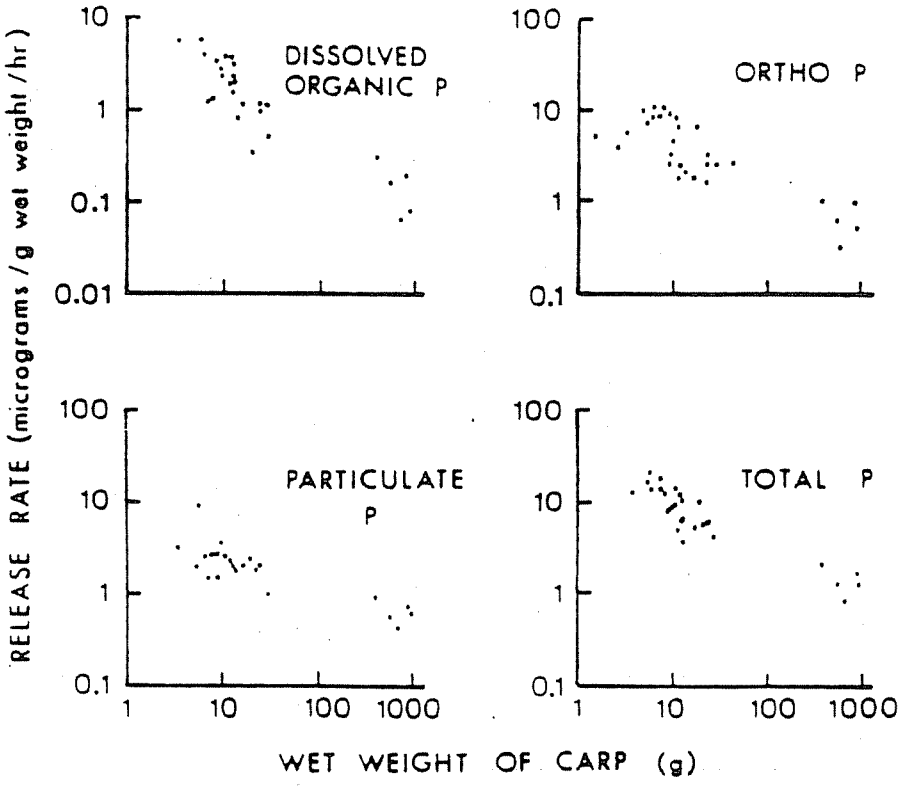

Figure 3. Release of various forms of phosphorus (P) by different-sized carp in 22° C water. Source: Lamarra 1975.

It may be concluded that in lakes infested with carp or bullheads, their removal by rotenone or other methods will substantially decrease phosphorus levels and thus algae levels. There are three complications which may amend this conclusion:

1) This same phosphorus can also be released from lake sediments in the absence of fish, particularly when the bottom becomes anoxic (Hutchinson, 1957). In lakes where this is a major source of annual phosphorus loading, the effect of bottom-feeding fish may be negligible. At least in Marion Lake, Smeltzer and Shapiro (1982) believed that excretion by carp and bullheads was by far the most important internal source of phosphorus.

2) Aquatic macrophytes such as *Elodea* also act as "phosphorus pumps" (Bandow, 1980; Welch, 1980) and these may proliferate in the absence of bottom fish which uproot and destroy them. This was the case in 14-foot deep Carls Lake, Minnesota (Bandow, 1980); after black bullheads were poisoned with rotenone, aquatic macrophytes (including *Elodea*) were no longer held in check, and expanded to occupy the entire lake bottom. These plants released large amounts of phosphorus and ammonia from the sediments, essentially negating the water quality benefits gained by killing the bullheads. Bandow did note, however, that in deeper lakes the growth of rooted aquatic plants should be less extensive.

3) When benthivorous fish are eliminated by any method which leaves their carcasses in the lake (e.g., rotenone), the phosphorus released by decay will at least temporarily mollify the beneficial effects of destroying them. For example, Smeltzer and Shapiro (1982) calculated that the phosphorus released by decaying carp and bullhead carcasses following rotenone treatment of Marion Lake would be equal to a third of the sediment phosphorus liberated by the fish if they were alive. As a consequence, phosphorus levels and algae would not decline as rapidly as expected following treatment. However, the authors pointed out that phosphorus release from fish decay constituted only a single "pulse" of loading, whereas excretion was a chronic source.

In summary, elimination of bottom-feeding fish with rotenone can be expected to lower phosphorous levels and thus algal abundance in lakes where:

1) algal abundance is limited by phosphorus; 2) lake sediments contain substantial amounts of phosphorus; and 3) potential sediment phosphorus released by fish is high compared to other mechanisms (e.g., sediment phosphorus release during periods of oxygen-depletion in the bottom muds).

The degree of phosphorus reduction may be roughly predicted from Lamarra's (1976) equations when estimates of fish biomass, size distribution and phosphorus loading from other source are available.

Effect of Decaying Fish on Phosphorus Levels - The phosphorus content of fish has been studied by several authors (Table B). Phosphorus made up between 0.3 and 1.6% of the whole-fish wet weight in these studies. While phosphorus content of fish varies somewhat with species, age, sex season, sexual maturity, and trophic state of the lake (Vinogradov, 1953; Dunst et al., 1974), Bull and Mackay (1976) suggested that an average value of 0.4% is adequate for a wide range of fish populations.

When fish die, the phosphorus bound in their carcasses must be broken down into the dissolved inorganic or organic form, mostly by bacterial action and autolysis, before it is usable by phytoplankton. This breakdown is extremely rapid; Wetzel (1983) states that "small fish" lose 7% of their total substance immediately upon death, and that 28% has been released within 24 hours under aerobic conditions in 20-25° C water. Once phosphorus is in an available form it is taken up so rapidly by algae and other plants that it is often not measurable. The release of this phosphorus from fish carcasses following rotenone treatment has been suggested as a cause of algae blooms (Funk and Moore, 1984).

Most fish killed by rotenone sink to the bottom of lakes undetected. It has been estimated that at 57-58° F (the average fall surface water temperature of lakes treated in Washington), only about 20-30% of the dead fish would surface within 24 hours. Thus, even when a concerted effort is made to recover all carcasses, at least 70% of the phosphorus content of the fish stock will be released into the lake through decay.

One final figure is necessary to estimate the amount of phosphorus (in g/ha) released by decaying carcasses: the total weight of fish per unit of surface area, or standing crop. This can vary considerably depending on the lake and the fish present. Bennett (1962) presented mean standing crop values for nineteen fish (usually in combination with other species) in U.S. lakes and reservoirs. Mean values for fish found in Washington state ranged from 4 lbs/acre (4.5 kg/ha) where trout dominated, to 100 lbs/acre (112 kg/ha) where carp dominated. The maximum standing crop recorded for U.S. waters was 1,235 lbs/acre (1,384 kg/ha) in Iowa ponds (Bennett, 1962; Dunst et al., 1974). Two Indiana reservoirs that contained a mixed population of warmwater species (mostly bullheads, bluegill, and carp) were rotenoned and then completely drained; this procedure provided standing crop figures of 153 lbs/acre and 300 lbs/acre (171 and 336 kg/ha) for the two lakes (Barry, 1967).

Table B. Phosphorus (P) content of fish.

Species	% of Wet Weight	Reference
Fish in general	0.3	Vinogradov (1953)
Atlantic salmon	0.168 a/	
Brown trout	0.246 a/	
Black crappie	0.7 b/	Burgess (1966)
Bluegill	0.8 b/	
Redear	0.6 b/	
Warmouth	0.5 b/	
Gizzard shad	0.6 b/	
Golden shiner	0.5 b/	
Brown bullhead	0.5 b/	
Longnose gar	1.6 b/	
Sockeye salmon (prespawning)	0.384	Donaldson (1967)
Sockeye salmon (spent)	0.345	
Rainbow trout	0.4	Bull and Mackay (1976)
Carp	0.5	
Northern squawfish	0.4	
Largescale sucker	0.3	

a/ Listed as phosphorus content of "soft part" of fish; may not reflect percentage of the whole fish.

b/ Burgess' figures originally reflected percentage of phosphates in the fish. Here his figures have been modified in accordance with Dunst et al. (1974), who reported percentage of phosphorus.

In Washington lakes proposed for rotenone treatment - often characterized by an out-of-balance fish population - total standing crop is often on the order of 75 lbs/acre (84 kg/ha); but where carp or goldfish dominate, this figure can be much higher (Fletcher, WDW, pers. comm.). For example, Picnic Point Pond in western Washington contained about 362 kg of goldfish (and almost no other fish) when rotenoned in 1980, yielding 228 kg/ha or 203 lbs/acre (calculated from data collected by University of Washington students and WDW biologist; Washington Department of Wildlife, 1981; and length-weight regressions for goldfish in Carlander, 1969).

Roughly, the decay of fish killed by rotenone could release as much as 0.3 kg P/ha into an out-of-balance mixed species water with 84 kg fish/ha for a carp or goldfish infested water with 300 kg fish/ha, the estimate jumps to 1.2 kg P/ha. These estimates are based on P = 0.4% of the wet weight of a fish.

In one respect, whatever the biomass of decaying fish and consequent phosphorus release, the phosphorus released by carcasses is phosphorus that would be released in any event when the fish die a natural death. On the other hand, in addition to the large biomass of "target" fish that are killed, there are always some residual trout left in the lake at the time of rotenone treatment. These are stocked fish, and thus the phosphorus in their carcasses represents an addition of phosphorus to the lake that occurs as a result of the trout program. Generally, however, the biomass of trout in a lake designated for rotenone treatment is small. These lakes are usually taken out of production as fry growing lakes the year of the treatment and given a nominal stocking of legal sized trout. Most of these fish are readily caught before the fall treatment. For example, 38% of the stocked legals in Pine Lake, Washington were taken by fishermen on Opening Day, 1980, and the majority removed within a week (Zisette, 1981). From a nutrient-loading standpoint, the essential difference between natural mortality and rotenone poisoning is that in the case of rotenone, all the phosphorus contained in the lake's standing crop of fish is released at the same time, rather than gradually.

There is no way to carry the estimate of how much of this phosphorus could become available for algae growth without some knowledge of a particular lake's limnology; too many factors influence the fate of phosphorus, of which the most important are:

1) Flushing rate of the lake . In rapidly flushing lakes, even high phosphorus loads can be insignificant (Welch, 1980). This may occur in some Washington lakes that are rotenoned in the fall, just prior to relatively massive rainfall and flushing. Naturally, the effect of sudden nutrient release in a lake with little outflow would be greater.

2) Conditions at the water-sediment interface . If the lake's hypolimnion is aerobic, much of the phosphorus released from the carcasses will be quickly tied up by metal complexes and resettle, unavailable for algae growth. Anaerobic conditions, on the other hand, would allow much of this phosphorus to reach the overlying water where it could be used by algae.

In many instances algae blooms occur shortly after rotenone treatment and some authors suggest that the release of phosphorus from decaying carcasses is a contributing factor. Table C shows the results of several studies where algal abundance was measured or noted shortly after rotenone treatment. In nine of eleven bodies of water, an algae bloom developed following rotenone treatment (although on Fern Lake, an application two years later produced no bloom). A "bloom" in this case is any increase in total algae (measured in chlorophyll a, cells/l, etc.) thought to be significant by the investigator(s).

As Table C shows, it is impossible to determine exactly why blooms occurred following rotenone. The two most likely hypotheses are phosphorus released from carcasses and/or a decrease in grazing following the annihilation of zooplankton, but it is impossible to separate the effects of the two. While no definintive answer exists, it is interesting to note that there were no fish in Burress' (1982) ponds, yet a bloom still developed following rotenone. Clearly, carcasses played no role in that case. Also, no bloom developed on Third Sister Lake (Brown and Ball, 1943a), zooplankton was only mildly affected by rotenone, cladocerans were never absent from the open water. These two examples seem to suggest that phosphorus released from fish carcasses is not nearly as important as reduced phosphorus grazing in causing algal blooms. Contradictory evidence from Carls Lake (Bandow, 1980) and Fern Lake 1962 (Fowler, 1973) - where no blooms developed despite the near annihilation of grazers - make firm conclusions impossible. Where they occur, it is likely that both phosphorus release from carcasses and reduced grazing are responsible for post-rotenone algae blooms, with the relative importance of each determined by the particulars of each lake.

Quantitative data are available from seven of the studies listed in Table C. These are graphed in Figures 4 through 10, showing the timing and magnitude of post-rotenone algae blooms where they developed.

Comparing "bloom" levels in a rotenon ear with the algae levels during that same period in a nonroter e year is perhaps the best way to gauge the magnitude of these b oms. These type of data are available for Pine Lake, Washing on and Hodges Reservoir, California. On Fern Lake, Washington, a continuous 12-year record of phytoplankton levels provides us with five seasons of data in nonrotenone years for comparison with the bloom that followed

Table C Response of phytoplankton shortly after rotenone treatment in selected lakes.

Name, location	Month of treatment	Length of study before treatment	Length of study after treatment	Did Algal bloom occur shortly after rotenone?	REASON FOR BLOOM: Increase in nutrients due to decay	REASON FOR BLOOM: Decrease in grazing by zooplankton	REASON FOR BLOOM: other	Comments	References
Pine Lake, western Washington	October	1 year, 3 months	2 months	yes	X	X		"...chlorophyll a increase... likely influenced by absence of grazing pressure." "Phosphorus remineralization of the decomposing organisms may have been at least partially responsible for an observed elevation in phosphorus."	-Zisette 1981 -Welch et al. 1981 -Municipality of Metropolitan Seattle 1981
Fern Lake western Washington	June	2 years	10 years	yes (1960) no (1962)		X		"slight increase in phytoplankton...occurred several weeks after the application of rotenone" in 1960.	-Kiser et al. 1963 -Fowler 1973
Bonham State Park Lake Texas	April	1 day	17 days	yes	X	X[a/]	X[b/]	"There was an increase in the number of [phyto-]plankton after addition of rotenone products."	Bonn & Holbert 1961
Lake Lavon (Price Creek Cove) Texas	June	unclear[c/]	unclear[c/]	yes	X	X[a/]	X[b/]	see above	Bonn & Holbert 1961
Patricia & Celestine Lakes Alberta, Canada	September	1 month	3 years, 5 months	yes				"Compared with subsequent years and prerotenone abundance, there was a small phytoplankton bloom shortly after treatment."	Anderson 1970

Table C Continued

Name, location	Month of treatment	Length of study before treatment	Length of study after treatment	Did Algal bloom occur shortly after rotenone?	REASON FOR BLOOM: Increase in nutrients due to decay	REASON FOR BLOOM: Decrease in grazing by zooplankton	REASON FOR BLOOM: Other	Comments	References
Ponds I & II Georgia	---	1 day	1 week	yes				"Substantial phytoplankton blooms developed within 3 days" [compared to control pond].	Burress 1982
Hodges Reservoir California	February	1 year, 1 month	3 weeks	yes				"Subsequent to treatment with Pro-Noxfish it was observed that a diatom pulse was developing."	Hoffman & Payette 1956
Carls Lake Minnesota	September	1 year, 5 months	2 years, 1 month	no				Although chl a and phytoplankton in general declined, author noted a "strong increase" of Melosira and Cryptomonas following rotenone, which he credited to decrease in grazing following the kill.	Bandow 1980
Third Sister Lake Michigan	May, August	1 year	1 year	no				"Most of the phytoplankton groups showed little or no change following the introduction of rotenone. A gradual decrease throughout the summer was noticeable for Chroococcales with an accompanying increase in diatoms... and Dinobryon.	Brown & Ball 1939

a/ authors actually gave reason as "reduction in plankton-feeding fishes," but none existed in the lakes; we assume they were referring to herbivorous zooplankton.

b/ "additional depth of light penetration" given as other reason for bloom.

c/ samples taken "at approximate 2-week intervals during the year resulted in a total of 180 analyses for 18 trips."

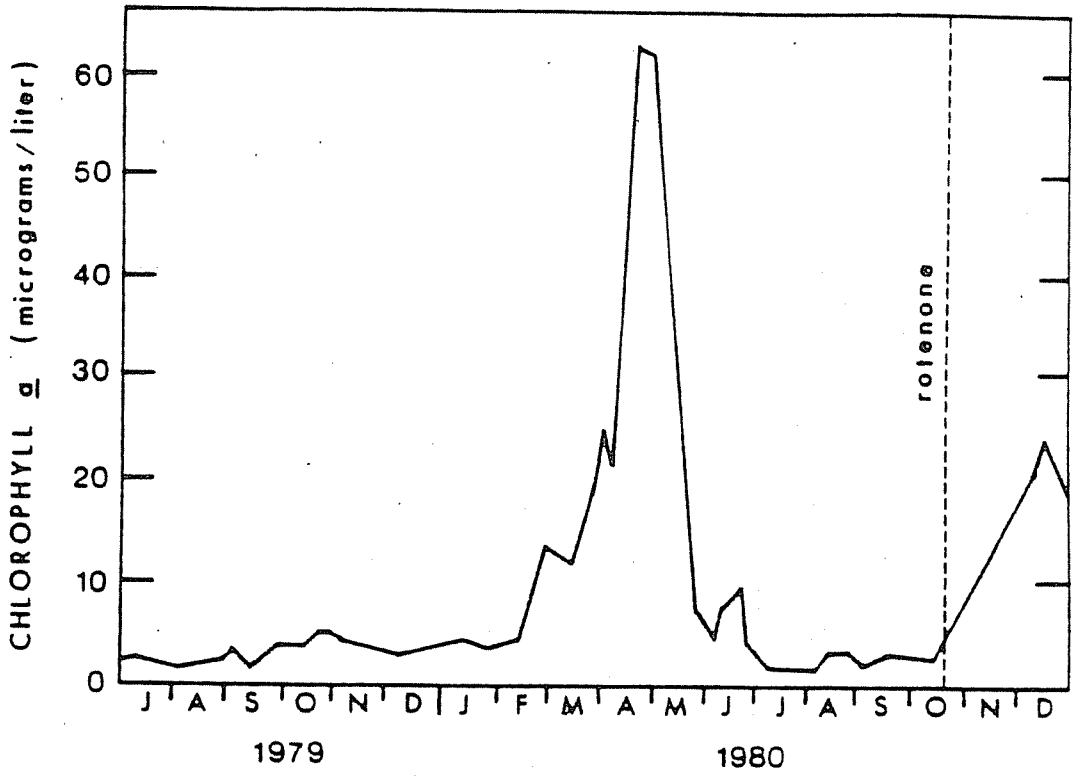

Figure 4 Volume weighted mean chlorophyll a content in the epilimnion of Pine Lake, Washington, before and after rotenone. Source: Welch et al. 1981.

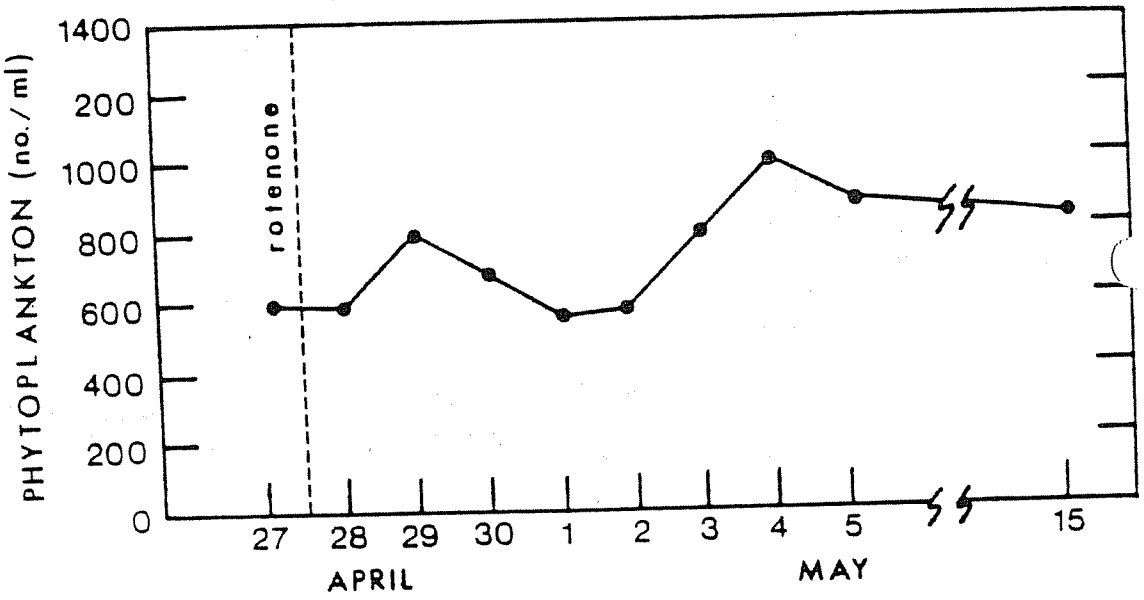

Figure 5 Phytoplankton levels in Bonham State Park Lake, Texas, before and after rotenone. Source: Bonn and Holbert 1961.

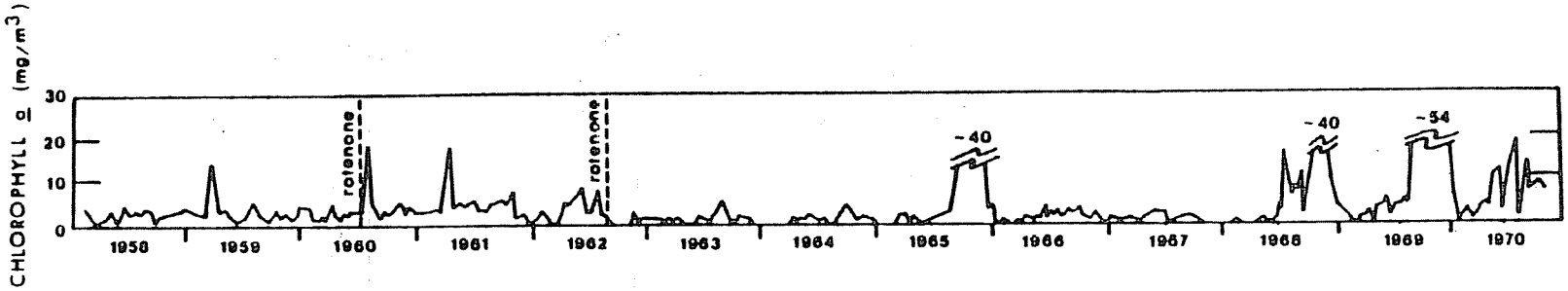

Figure 6 Abundance of algae (as chlorophyll a) in Fern Lake, Washington over a twelve-year period. Rotenone was applied in 1960 and 1962. Artificial fertilizers were applied in 1965,1968, and 1969. Source: Fowler 1973.

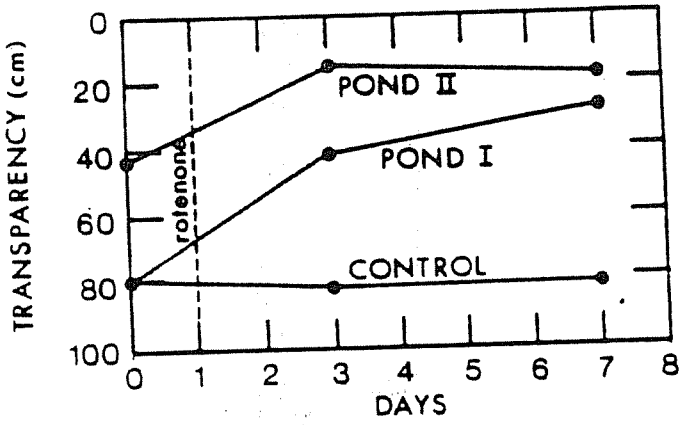

Figure 7. Secchi disc transparency in two ponds treated with rotenone, and one untreated control pond. Source: Burress 1982.

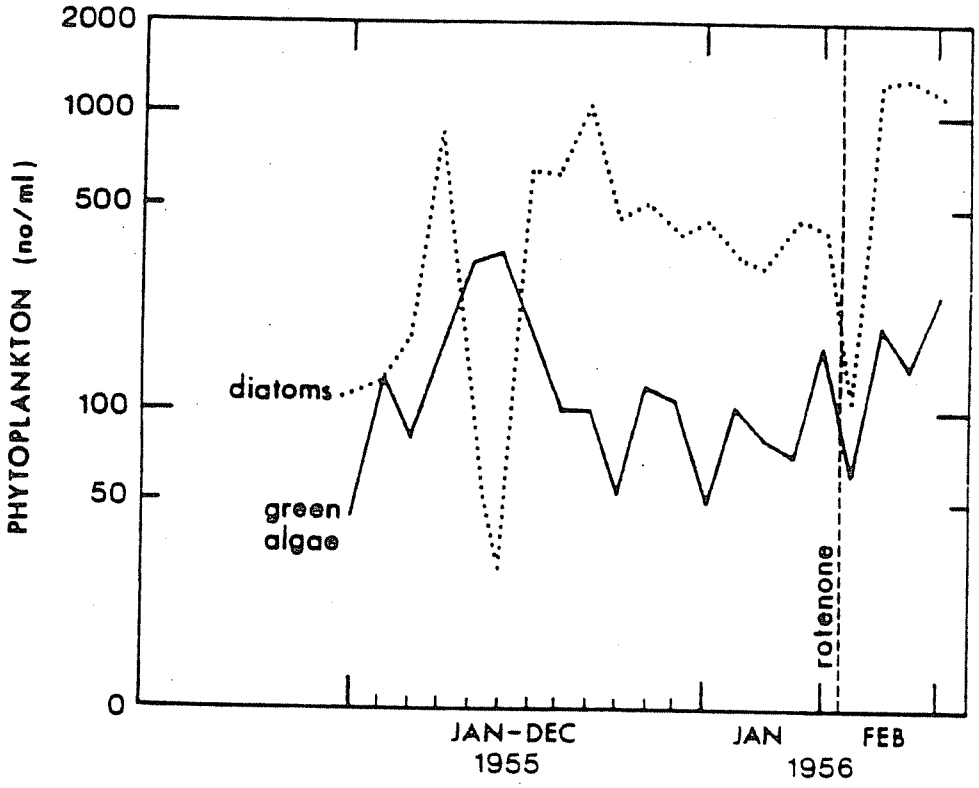

Figure 8. Diatom and green algae levels in Hodges Reservoir, California, before and after rotenone. Source: Hoffman and Payette 1956.

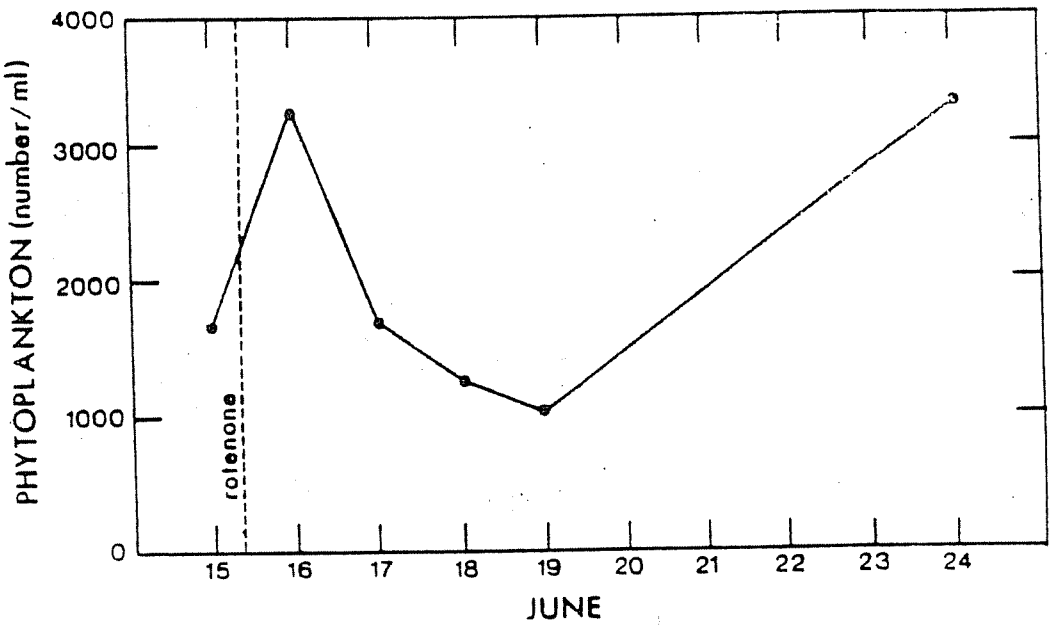

Figure 9. Phytoplankton levels in Lake Lavon (Price Creek Cove), Texas, before and after rotenone. Source: Bonn and Holbert 1961.

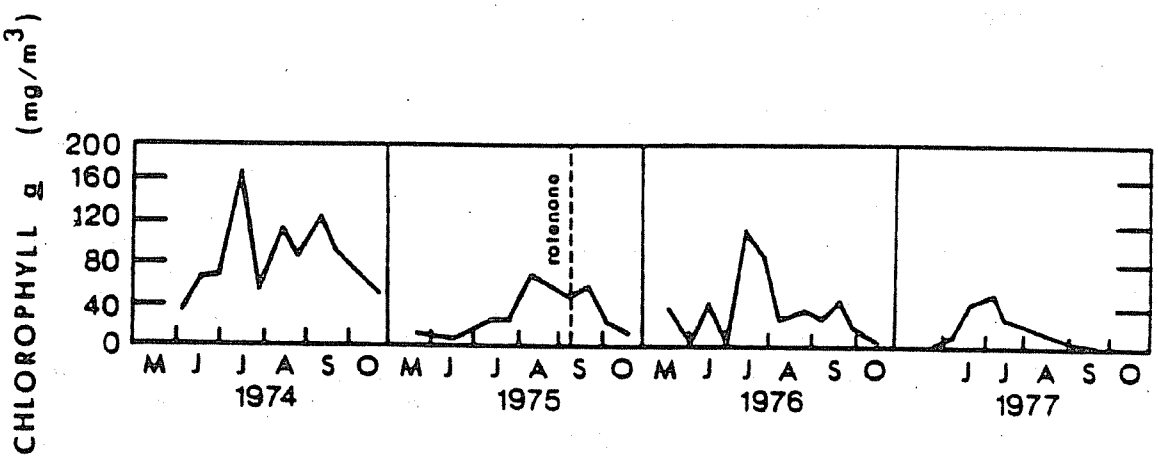

Figure 10. Phytoplankton levels (as measured by chlorophyll a) in Carls Lake, Minnesota, before and after rotenone. Source: Bandow 1980.

the rotenone treatment in 1960 (artificial fertilization and a second rotenone treatment obscure results in the other years).

The algae levels in rotenone and nonrotenone years are shown for these three lakes in Table D. Algae levels increased 4 to 6 fold shortly after rotenone treatment, when compared with "normal" levels in nontreated years. It is important to remember that blooms do not always occur following rotenone, even in a particular lake (i.e., Fern Lake). Table D demonstrates the magnitude of post-rotenone blooms where they occurred. These blooms generally lasted 1-2 months, judging by the studies on Pine, Fern and Hodges lakes. Most rotenone applications in Washington take place in the fall; a bloom, if one occurs, would be expected to subside sometime in December with decreasing sunlight and flushing, as was the case on Pine Lake in 1980.

There is little information whether the increased phosphorus from fish carcasses decaying on the bottom will cause algae blooms (or intensify regularly-occurring blooms) the following season. Algae levels in Fern Lake in 1961 and 1963 (following the 1960 and 1962 treatments) indicate nothing out of the ordinary when compared to other years. These data agree with the wealth of information available from lakes and ponds that have been artificially fertilized; Shapiro (1970) cited the early work of Einsele and Edmondson, noting that "single-shot" fertilization with superphosphates was ineffectual - the results lasted only for the year of application and did not carry over to any extent to the next year unless the fertilization was repeated. More recent experiments in Canada have confirmed this: when Schindler and Fee (1974) fertilized a lake with phosphorus, algae increased dramatically during the two treatment years, but fell to pretreatment levels as soon as phosphourus input was curtailed. Figure 15 shows the same pattern in Fern Lake, which was fertilized in 1965, 1968 and 1969. This is generally true of lakes in the low or medium productivity range (Wetzel, 1983). In eutrophic lakes this fertilizer might be recycled from the sediments, causing further blooms.

There is an essential difference between artificial fertilization and phosphrus released from rotenone killed carcasses: artificially fertilized lakes (including the great many eutrophic lakes that recieve phosphorus from septic tanks, runoff, etc.) are enriched by phosphorus from external sources. In a rotenoned lake, the sudden enrichment comes from fish that obtained all their phosphorus from the lake. Thus, there is no net increase in phosphorus, only a sudden and unusual availability that takes place following the poisoning. This essentially confines the bloom potenial to the year of treatment, even in eutrophic lakes. For example, researchers concluded that on culturally-eutrophic Pine Lake, "phosphorus remineralization of decomposing organisms (following the 1980 rotenone treatment) may have been at least partially responsible for an observed elevation in whole-lake phosphorus

Table D. Magnitude of algal blooms following rotenone treatment in some test waters. Values are approximations only, in some cases based on interpretation of graphic data. Blooms on these lakes lasted 1-2 months.

Lake, year	Method of comparison	Unit	ALGAE LEVELS Nonrotenone year(s)	Rotenone Year	% Increase	Reference
Pine Lake western Washington 1980	means of November and December chl a levels in 1979 (no rotenone applied) and 1980 (rotenone applied on October 23).	ug chl a / l	3.5	18.5	429%	-Zisette 1981 -Welch et al. 1981
Fern Lake western Washington 1960	means of peak chl a levels nearest the end of June in 1958, 1959, 1961, 1963, and 1964 (no rotenone applied) with peak in 1960 (rotenone applied on June 4).	mg chl a / m^3	13.4	59.7	346%	Fowler 1973
Hodges Reservoir California 1956	mean diatom level in February 1955 (no rotenone applied) with February 1956 (rotenone applied January 31).	#/ml	125	730	484%	Hoffman & Payette 1956

(about 5 micrograms/l while inflow phosphorus was negligible). Available data, though inconclusive, suggest that existing autumn rotenone applications do not have a significant impact on annual phosphorus dynamics and algal blooms in Pine Lake" (Municipality of Metropolitan Seattle, 1981).

As shown in Figure 1, there is a second way in which decaying fish carcasses have the potential for increasing phosphorus levels: decomposition requires oxygen in most cases, and large numbers of fish carcasses may turn a lake bottom anaerobic. Sediment phosphorus is normally trapped (or release is insignificant) in aerobic bottoms where the overlying water contains more than 1 mg oxygen per liter (Wetzel, 1983). But when oxygen levels fall below this point, redox potentials also decrease, and a sudden release of phosphate phosphorus occurs from the sediments. When the water is reoxygentated, phosphate phosphorus is again resettled and trapped in the sediments. In this case, the source of phosphorus is not the fish carcasses themselves, but the lake sediment that has become depleted of oxygen by their decay. Phosphorus released from the sediments by any means can be a major cause of algae blooms (Cook et al., 1977).

Almost no data are available from rotenoned lakes on this subject. While a number of studies routinely recorded oxygen levels at various depths before and after treatment, there is often no mention of whether or not the fish carcasses were removed from the study lake or allowed to decay. In others, such as Pine Lake, (Welch et al., 1981), the bottom was anoxic even in nonrotenone years.

In research designed to determine the indirect effects of rotenone on municipal water supplies, Bonn and Holbert (1961) poisoned two Texas lakes, killing 79 pounds of fish per acre on one and 145 lbs/acre on the other. After a weigh-in, the body cavities of the fish were punctured and they were scattered back into the lakes to create a natural post-kill condition. Oxygen levels as well as other chemical and biological parameters were measured at various depths, including the bottom. Water temperatures were high (70-91° F) and the carcasses decayed rapidly, with significant increases in total organic nitrogen and bacterial levels. Although some oxygen must have been consumed by the bacterial decomposition, Bonn and Holbert reported no significant change in oxygen levels. A "bloom" did develop in both lakes, but not because of anaerobic phosphorus release from the sediments. However, these Texas lakes were fairly shallow (maximum depths of 15.5 and 9 feet), and oxygen depletion in deeper lakes, where oxygen diffusion from the surface takes more time, might still occur.

The hypolimnia of many lakes - especially eutrophic ones - typically become anoxic during the summer and winter (Welch, 1980). Where this yearly pattern occurs, rotenone-killed fish carcasses cannot be expected to aggravate sediment phosphorus release, since

oxygen levels are already below 1 mg oxygen/liter. In culturally eutrophic Pine Lake, for example, the October 1980 rotenone treatment occurred well after the bottom became completely anoxic in early July; as in other years, the lake turned over in early December and the bottom was reoxygènated (Welch et al., 1981).

Effect of Trout Stocking on Phosphorus Levels - When a fingerling trout is stocked in a lake, the 0.4% of its wet weight that is phosphorus is added to the system. When and if the trout is caught by an angler, this amount of phosphorus plus 0.4% of the added weight that the trout has gained during its growth period in the lake is removed.

Phosphorus tied up in the tissue of living fish is not immediately available for use by algae; but it is quickly released back into the water if the fish dies in the lake. In the same way, a fish removed from the lake represents a loss from the total phosphorus pool and ultimately from the available phosphorus pool.

Fish stocking records and catch estimates can be used to determine if the process of stocking and harvesting trout fertilize lakes or reverse fertilization. While fish stocking records are readily available for all of Washington's stocked lakes, creel survey estimates of season-long catch are more difficult to come by. Where season-long catch estimates are not available, catch estimates from opening day of lowland fishing season (usually occuriing on the third or fourth Sunday in April) can be used as a minimum value for phosphorus removal by angling. Creel surveys are performed on all of Washington's lowland trout lakes on opening day, and the statistical methodology for estimating catches of fingerling-origin trout on these lakes is well developed (Brown, 1978).

Table E displays the estimates of phosphorus added and removed by trout stocking and harvest on selected Washington lakes. All are lowland "trout-only" waters, most have been treated with rotenone, and they are fairly representative in terms of stocking and catch rates. These particular lakes were selected because reliable season long (or opening day) catch estimates were available.

Only the Kitsap County lakes surveyed by Johnston (1973) showed a net gain in phosphorus; Table E indicates that in most cases, more phosphorus is removed from trout-only lakes than is added. The amount varies considerably, however, mostly depending on the percentage of the fingerling introduction that is caught. In eastern Washington lakes, this percentage is typically high, while in western Washington it is usually much lower. This difference is due mostly to fingerling survival: mark-recapture studies have shown that survival from stocking to opening day ranges from 2% to 61% in western Washinngton lakes and from 70% to 87% in eastern Washington lakes (Washington Department of Wildlife, 1968).

Table E Estimated annual amounts of phosphorus (P) added and removed by fingerling trout stocking in selected Washington lakes.

Name Year(s) [a/] Reference	Quincy Lake 1981 Jackson 1983	Susan Lake 1981 Jackson 1983	Lower Hampton 1981 Jackson 1983	Upper Hampton 1981 Jackson 1983	Liberty Lake 1978 WDG files	Lake Morton 1974 Cummins 1975	Martha Lake [b/] 1978, 1980 WDG files	Various lakes in Kitsap Cty. 1972, 1973 Johnston 1973	Pine Lake 1979 WDG files
fingerling stocking rate (fish/acre/yr)	610	607	668	225	387	377	463	400	454
fingerling size (fish/lb)	130 [c/]	130 [c/]	130 [c/]	130 [c/]	80	110	170	128	150
fingerling stocking rate (kg/ha/yr)	5.26	5.23	5.76	1.94	5.42	3.84	3.05	3.50	3.39
P added by fingerling stocking (g/ha/yr) [d/]	21	21	23	8	22	15	12	14	14
Survey period	season long	season long	season long	season long	season long	season long	Opening Day	Within 3 weeks of Opening Day	Opening Day
% of fingerling plant caught	45.3	37.9	65.3	45.8	60.0	21.0	5.4	1.8	3.0
catch (fish/ha/yr)	541	568	1078	256	574	196	62	18	34
catch (kg/ha/yr) [e/]	162	170	323	77	86	27	9	3	5
P removed by fishery (g/ha/yr) [d/]	648	680	1292	308	344	108	36	12	20
Δ P (g/ha/yr)	-627	-659	-1269	-300	-322	-93	-24	+2	-6
P load (g/ha/yr)					3700 [f/]				4146 [g/]
P removal as a % of P load					8.70%				0.14%

a/ refers to harvest year - fingerlings were planted the previous year.

b/ mean of both years' data.

c/ average size for these lakes (Joe Foster, WDG biologist, personal communication).

d/ assumes that P = 0.4% of a trout's wet weight (Bull and Mackay 1976).

e/ weight/fish = 0.14 kg for lakes in Kitsap County, and lakes Morton, Martha, and Pine, based on average Opening Day fork length of 8.75 inches (Johnston 1973) and length/weight regressions in Carlander (1969). Weight/fish ≈ 0.15 kg for Liberty Lake, based on average Opening Day size (John Hisata, WDG biologist, personal communication). Weight/fish ≈ 0.3 kg for lakes Susan, Quincy, and Hampton, based on Opening Day size of 11.5-12.0 inches (Jackson 1983) and regressions in Carlander (1969).

f/ 1974-78 prerestoration figures (Ron Pine, D.O.E., personal communication).

[illegible] 1981

Table E shows that net removal of phosphorus in the eastern Washington lakes varied between 300 and 1,269 g/ha/yr. In the western Washington lakes, net removal ranged from 6 to 93 g phosphorus/ha/yr., with a net addition of 2 g of phosphorus/ha/yr in the Kitsap County lakes. While most of the lakes in western Washington are represented by only Opening Day or partial season catch estimates, it is well documented that a major portion of the season long catch occurs on Opening Day (Johnston, 1973; Cummins, 1975).

Phosphorus-loading data are available for only two lakes, Liberty Lake and Pine Lake. Both of which are culturally eutrophic lakes, suffering from nuisance algae blooms, and both are phosphorus limited, making analysis of the significance in terms of the lakes' total phosphorus budgets difficult.

In the case of Pine Lake, Table E makes it clear that the net phosphorus removed by trout harvest (6 g/ha/yr) is insignificant when compared to the total phosphorus loading of 4,146 g/ha/yr. Since no phosphorus budgets exist for the other western Washington lakes shown in Table E, it can only be assumed that they lie within the broad range of values suggested by Vollenweider (1968): between 700 and 2500 g/ha/yr on low nutrient lakes, and between 1300 and 5000 g/ha/yr on "problem lakes". It is easy to see that either addition or removal of phosphorus in the range given for western Washington lakes in Table E (+2 to -93 g/ha/yr) is negligible compared to the phosphorus loads.

The situation is different in eastern Washington lakes, where phosphorus was removed by anglers in much larger amounts - 300 to 1,269 g phosphorus/ha/yr. These withdrawals of phosphorus from lakes could play an important role in counteracting the eutrophication process. On Liberty Lake, for example, anglers removed 322 g/ha in 1978. Total phosphorus loading in 1974-1978 was 3,700 g/ha/yr so that 8.7% of the phosphorus added to the lake in 1978 from all sources could have been removed by anglers that year.

Bull and Mackay (1976) drew a similar table for two Canandian lakes: one eutrophic, the other oligotrophic - and concluded that even at the maximum sustainable yield (MSY), less than 1% of the phosphorus entering the lakes could be removed by anglers. Thus, fish harvest could neither slow nor reverse the process of eutrophication. While this conclusion agrees closely with our analysis of the western Washington lakes, it contradicts the eastern Washington data. The reason for the difference is that Bull and Mackay estimated the top catch from their lakes was less than 3 kg/ha/yr, an extremely low figure that compares only with the Kitsap County lakes in Table E. A likely explanation for this low estimate is that the Canadian lakes were not sustained by

hatchery stocks; a much lower catch-per-unit-surface-area would be expected from a natural population of salmonids than from a stocked lake.

Burgess (1966) noted that sport harvest of warmwater fish removed over 5,000 lbs. of phosphates from Lake Harris, Florida, over a 15-month period. The author likened this to removing all the phosphates from the annual untreated waste of over 1,000 persons. He further noted that while phosphorus removed by anglers would not cause an immediate reduction of nutrients in the lake, it would serve as a deterrent to eutrophication. Dunst et al. (1974) concurred that harvesting fish might significantly reduce nutrients in some problem lakes.

While fishing is not a consequence of a "trout-only" program, the large catches associated with eastern Washington "trout-only" lakes do not ordinarily occur in waters managed for "mixed-species". On the "trout-only" waters in eastern Washington cited in Table E, for example, the average harvest was 1,491 trout/acre/year; the average on four similar-sized "mixed-species" lakes (two in eastern and two in western Washington) was only 120 "warmwater" fish/acre/year. Trout catches were not included in the latter calculation because trout planted as "legals" just before Opening Day do not constitute a net loss of nutrients when caught. These figures indicate that harvest (and thus nutrient removal by anglers) is roughly ten times greater on high-yield "trout-only" lakes than on "mixed-species" lakes.

Effects of Rotenone on Trout Stocking and Grazing - Figure 1 shows that both rotenone treatment and subsequent trout stocking affect grazing by zooplankton.

Poisoning a lake with rotenone temporarily destroys the zooplankton and thus decreases grazing (Figure 1). 95 to 100% of the open-water zooplankton are destroyed within a few days, and crustacean plankters are generally absent from open-water tows for two to twelve weeks. Since these are the most important grazers, a decrease in grazing followed by a surge in phytplankton could logically be expected.

Algae blooms commonly follow rotenone treatments and reduced grazing is often cited as a cause (Table C). Phosphorus released by carcasses probably also contributes to these blooms, and it is impossible to separate the effects of the two. Long-term (i.e., beyond the year of application) effects of rotenone on grazing are unlikely; in most cases, zooplankton have recovered in abundance and diversity to prerotenone levels within two to twelve months after treatment.

Zooplankton populations eventually recover from rotenone poisoning, and they usually do so before fish re-enter the lake or are restocked. Figure 1 shows that in the absence of fish, large

zooplankters such as *Daphnia pulex* usually dominate the recovered community, and that they will continue to dominate if fish are not restocked or otherwise re-enter the lake. Also, already common plankters often increase in body size when they recover in the absence of fish.

These large zooplankters are more efficient grazers than small ones (Kerfoot, 1980). Burns (1969) found that the filtration rates of several *Daphnia* species were roughly proportional to the square or even cube of their body lengths. This difference in grazer body size may be even more important than grazer population size in controlling algae. Hrbacek et al. (1961) found that algae levels in a rotenoned Czechoslovakian backwater decreased despite a smaller standing crop of zooplankton, the reason being that a large daphnid suddenly became dominant following the fish kill.

This sequence - large grazers becoming dominant in the absence of planktivorous fish and reducing algae levels - has been repeatedly demonstrated in both small-scale enclosure experiments (Lynch and Shapiro, 1981; Andersson et al., 1978 and whole-lake situations (Shapiro, 1979). Reducing algae levels by increasing large grazer populations (either by killing planktivorous fish with rotenone or by introducing large piscivorous fish which accomplish the same thing) is one of the cornerstones of the "biomanipulation" movement, and the literature is abundant and detailed (i.e., Shapiro et al, 1982; Goad, 1982).

Shapiro and Smeltzer (1982) reviewed data from 13 Minnesota lakes poisoned with rotenone or toxaphene; seven showed transparency increases following poisoning, two probably became clearer, and four showed no change. Unfortunately, the species of "undesirable" fish eliminated - presumably planktivorous in at least some of the improved lakes - were not mentioned; these lakes were managed for bass, pike and walleye. The authors made the reasonable assumption that the lakes that cleared did so because of reduced algae levels, and that either increased grazing in the absence of fish (or both) was the cause.

Eutrophic Wirth Lake, Minnesota was intensively studied by Shapiro (1982) before and after rotenone treatment. The fish population, made up of crappies (*Pomoxis spp.*) (50%), bluegill sunfish (*Lepomis macrochirus*) (25%), carp (*Cyprinus carpio*) (15%) and perch, bullheads (*Ictalurus spp.*), suckers (Catostomidae), northern pike (*Esox lucius*) and largemouth bass (*Micropterus salmoides*) (10%), was poisoned in September 1977. The zooplankton population was virtually wiped out by the rotenone, but partial recovery was apparent 25 days later. Due to complications, it is impossible to tell whether or not a post-rotenone bloom occurred during this period. The following year a huge population of large daphnids (*D. pulex*) appeared, and algae levels decreased dramatically with a concurrent increase in transparency (Figure 11). The *Daphnia* population averaged 256,000 per m^2, or 32 individuals per liter, a

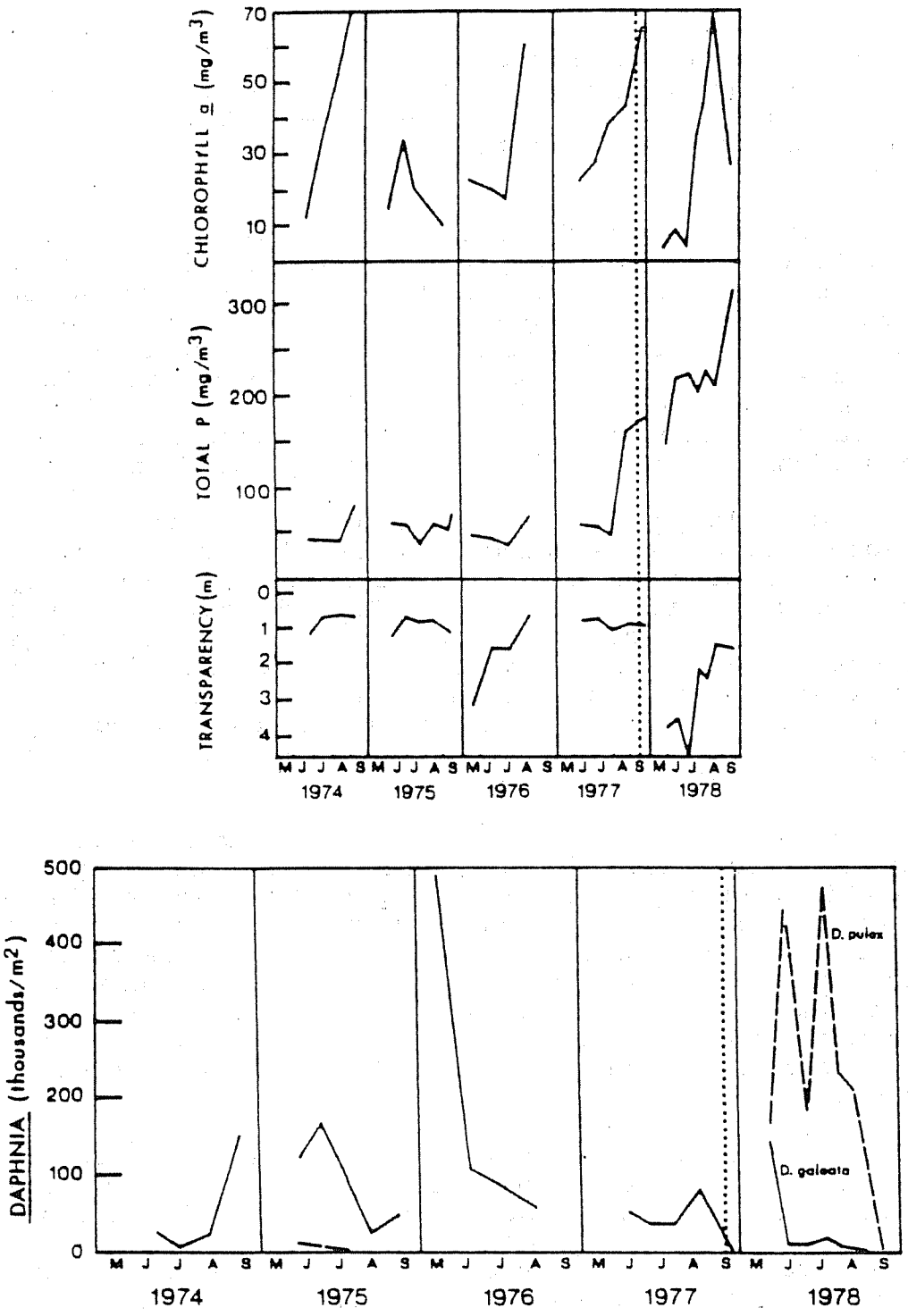

Figure 11. Algae (as chlorophyll a), phosphorus (P), transparency, and Daphnia in Wirth Lake, Minnesota. Dotted vertical line indicates date of rotenone treatment. Phosphorus levels in 1978 were increased by mechanical circulation, not through any biological process. Source: Shapiro et al. 1982.

concentration theoretically capable of removing virtually all filamentous blue-green algae from the lake (Lynch and Shapiro, 1981).

Within a short time, the blue-greens *Aphanizomenon, Oscillatoria* and *Anabaena* were reduced to low levels and replaced by green algae. Overall, algae levels in early 1978 were much lower than in the four years before rotenone, and Shapiro credited the large zooplankters. What makes Wirth Lake a particularly interesting case is that phosphorus levels were dramatically increasing (as a result of mechanical water circulation) during the same time that rotenone treatment and large daphnid increases were taking place; thus, algae levels decreased due to grazing despite "fertilization". However, later in 1978, chlorophyll levels increased and transparency decreased due to certain complications.

Clear Lake, Minnesota provides another example: Smeltzer (1982) noted that algae levels declined sharply the year following rotenone, and he credited increased grazing by large zooplankton in the absence of fish. Based on these and other experiments, Shapiro and Smeltzer (1982) concluded that "in balance, then, it would appear that use of fish toxicants does reduce algal abundance and that in some cases the effect has been longlasting".

Grazing Effects on Different Algae - Generally, when large grazers become dominant algae levels decrease, although there are some important exceptions to this pattern:

1) not all algae can be eaten by zooplankton;
2) some algae, though eaten, can pass through a zooplankter's gut unaffected; and
3) some algal species increase in number or change to inedible forms when grazing on them increases.

In the first two cases the net result is that algae levels are unaffected by increases in the number of grazers. In the third case, increased grazing may actually reverse the usual pattern and cause algal levels to increase. The dotted line in Figure 1 leading to an "*Aphanizomenon* flake bloom" depicts this pathway; it is a dotted rather than a solid line to indicate that it is an exception that only seems to occur under special circumstances.

Table F lists some algae that are not significantly affected by zooplankton grazing. In most cases, these forms are too large for plankters to eat. In some cases, gelatinous green algae like *Sphaerocystis* are eaten but pass intact through the grazer's gut. In still other cases, algal species secrete something that causes grazers to reject them, or that is actually toxic to zooplankton. The net result is the same: if these forms are dominant in a lake, grazing by itself can rarely be expected to reduce their abundance.

Table F. Algal types unaffected and suppressed by grazing. Numbers in parentheses refer to references in the literature (see below).

USUALLY UNAFFECTED BY GRAZING	USUALLY SUPPRESSED BY GRAZING
BLUE GREEN ALGAE: *Anacystis nidulans* (1)	BLUE GREEN ALGAE: *Chroococcus limneticus* (5)
Merismopedia sp. (1)	BLUE GREEN ALGAE: *Aphanizomenon flos-aquae* (2) b/
Synechocystis sp. (1)	*Cryptomonas* (5)
Gloeocapsa alpicola (1)	*Rhodomonas* (5)
Microcystis aeruginosa (2, 3)	*Cyclotella comta* (5)
Oscillatoria rubescens (4) a/	*Asterionella formosa* (5)
Oscillatoria agardhii (4) a/	*Oocystis lacustris* (5)
Anabaena flos-aquae (5, 1)	*Chlamydomonas* (1, 5)
Anabaena affinis (5)	*Coelosphaerium dubium* (2)
Anabaena sp. (6)	*Ankistrodesmus falcatus* (1)
Anabaena ("some species") (2)	*Chlorella vulgaris* (1)
Gloeotrichia sp. (6)	large diatoms (5)
Lyngbya sp. (6)	flagellates (5)
Synechococcus elongata (1)	euglenids (5)
Aphanizomenon sp. (7) b/	ciliates (5)
Synechococcus cedrorum (1)	
Mallamonas caudata (5)	
Peridinium willei (5)	
Cosmarium depressum (5)	
Sphaerocystis schroeteri (5)	
Elakatrothrix gelatinosa (5)	

(1) Arnold (1971)
(2) Sorokin (1968)
(3) Lampert (1981)
(4) Edmondson and Litt (1982)
(5) Porter (1973)
(6) Edmondson (1957)
(7) Lefevre (1950)

a/ *Oscillatoria* apparently inhibits only *Daphnia* sp.

b/ note that this species is listed in both columns; although Lefevre does not actually mention it, he probably was referring to the "flake" colony form of *Aphanizomenon* as inedible. Sorokin and others have found *A. flos-aquae* in the filementous or non-flake form to be readily eaten. See discussion on this species in text.

Many of these inedible forms of algae are the very ones responsible for obnoxious blooms in eutrophic "problem" lakes. Blue-green algae are usually the most objectionable, and as shown in Table F many of these are unaffected by grazing.

This has far-reaching implications for "biomanipulators" interested in cleaning up problem lakes. For example, Goad's (1982) proposal to increase grazing in Green Lake, Washington by eliminating yellow perch (*Perca flavescens*) and other planktivores was dismissed by Perkins (1982) because the problem-causing algae there were primarily *Gloeotrichia* and other inedible blue-greens. Unless the phytoplankton community could be changed to more edible forms - and there are ways to do this - no great improvements would likely occur.

Not all blue-greens are inedible, however. *Aphanizomenon*, for example, especially *A. flos-aquae*, is commonly found in many eutrophic and mesotrophic lakes in Washington; Liberty Lake and Lake Washington being two examples. *Aphanizomenon* has the ability to exist in two forms: when filamentous, it is readily eaten, especially by large grazers, and is a good food source (Sorokin, 1968). But it also can clump together in the "flake" or "grass-blade" form, which is inedible. Interestingly, this "flake" form often develops in lakes with abundant large grazers such as *Daphnia pulex* or *D. pulicaria*, apparently in response to heavy grazing pressure (Hrba'cek, 1964; Lynch, 1980; Shapiro, 1979; Bandow, 1980). This phenomenon occurs on a wide variety of lakes (Shapiro, 1979), and Lynch (1980) substantiated the fact that the large matted colonies of *Aphanizomenon* appears when *D. pulex* became abundant, and disappeared when the large grazers were eliminated by fish. Straskraba and Straskrabova' (1969) likewise reported that "extraordinarily high numbers of fish (reducing the grazing population) decreased the *Aphanizomenon* blooms" in a Czech reservoir.

But "flake" blooms of *A. flos-aquae* are not inevitable with abundant large *Daphnia*. Lynch (1980) found that "flake" blooms occurred only when the lake bottom was oxygenated. Shapiro (1979) showed that this was the case in a wide range of lakes: both large daphnids and oxygenated hypolimnia were necessary for such a bloom.

This, then, is a special case in which increased grazing by large zooplankters actually promotes nuisance algae blooms. There is yet another complication with *Aphanizomenon* blooms: since the "flake" or "grass-blade" form is clumped, water transparency in a lake usually increases rather than decreases. This leads to the paradox of high algae and chlorophyll a levels, but clear water at the same time. Edmondson (1980) noted that when large colonies of *Aphanizomenon* and *Anabaena* formed in Lake Washington in the presence of large *Daphnia*, the water "looked crumby but clear". While Lake Washington was enjoying the clearest water ever recorded, residents on the downwind side of the lake were

complaining about the huge matted colonies of blue-green algae (Litt, UW, pers. comm.; Shapiro, 1979). Because of this problem, Shapiro cautioned against using any technique to increase large *Daphnia* in a lake unless it could be ascertained that *Aphanizomenon* would not become dominant.

Grazing After Fish Are Restocked - Figure 1 shows that when trout are restocked in a rotenoned lake, the large grazers that developed in the absence of fish are again cropped back. Additionally, Figure 10 shows that when the large plankters are cropped back, a reduction in grazing can be expected, since the small grazers are less efficient at filtering algae. This should lead to an increase in algae.

There is a great deal of literature suggesting that when trout or other planktivorous fish are introduced into a formerly fish-free environment dominated by large grazers, the following things happen:

1) large grazers are reduced, or even eliminated entirely;
2) small grazers take their place;
3) grazing on algae is reduced as a result; and
4) algae levels increase, often dramatically.

Evidence comes in part from enclosure experiments where planktivorous fish have been added to plastic bags suspended in lakes. Lynch and Shapiro (1981) demonstrated that algal biomass increased and transparency decreased as they added more planktivorous fish to enclosures containing *Daphnia* (Figure 12). Andersson et al. (1978) also noted dramatic increase in algae when they added fish to enclosures in two Swedish lakes (Figure 13). Since they used fish that were both planktivorous and benthivorous, some of the increased algae was due to higher phosphorus levels. In both of the experiments above, the algae produced in the presence of fish were mostly edible blue-greens.

Similar studies in natural, whole-lake situations are rare. While a number of authors have investigated the effect of trout introductions on grazer populations in fish-free lakes (Kitchell and Kitchell, 1980; Anderson, 1972; Galbraith, 1967; 1974), almost none have extended their studies to include the phytoplankton.

Medical Lake in eastern Washington is an exception, with the fish, zooplankton, and algae extensively studied since 1974 (Scholz et al., 1985; Mires et al., 1981; Knapp and Soltero, 1983; Soltero et al., 1981). Medical Lake was treated with alum in 1977 to reduce phosphorus levels and clear the lake of nuisance algae. The lake responded with a change from blue-green to green algae, reduced algae levels, greater water clarity, and a predominance of large *Daphnia pulex*. The enhanced water quality prompted WDW to stock the lake - which previously could not support trout - with rainbow fingerlings beginning in 1978. These trout began feeding almost

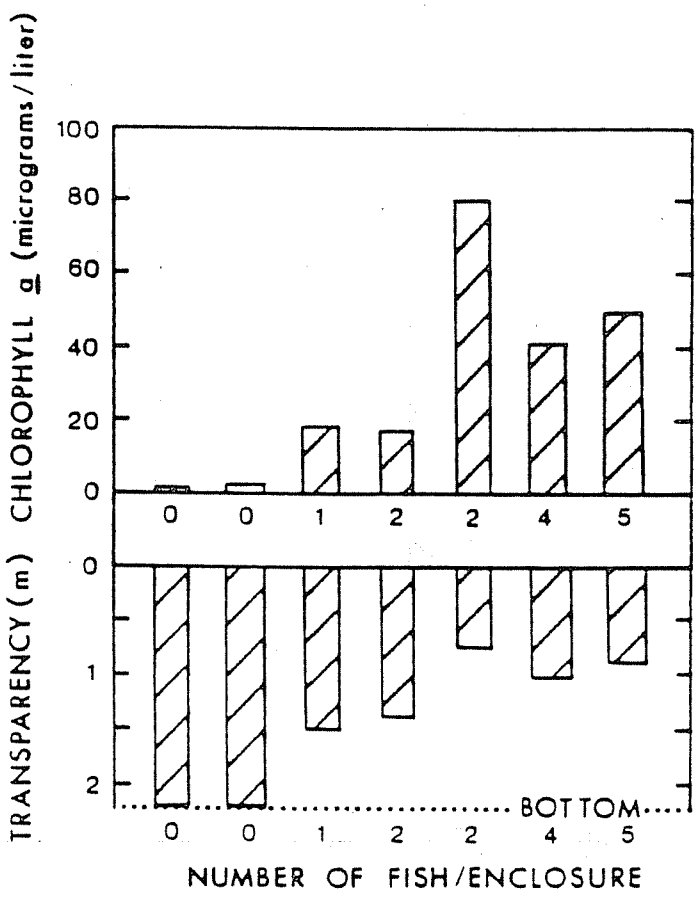

Figure 12 The effects of adding various numbers of zooplanktivorous fish to enclosures of lake water containing Daphnia. Source: Lynch and Shapiro 1981; Shapiro 1979.

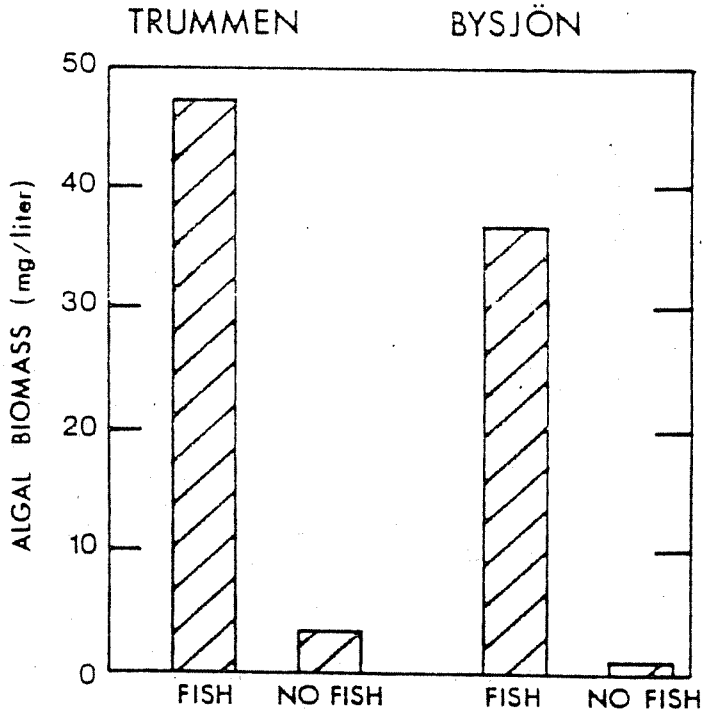

Figure 13 The effects of adding zooplanktivorous and benthivorous fish to enclosures in two Swedish lakes. Source: Andersson et al. 1978.

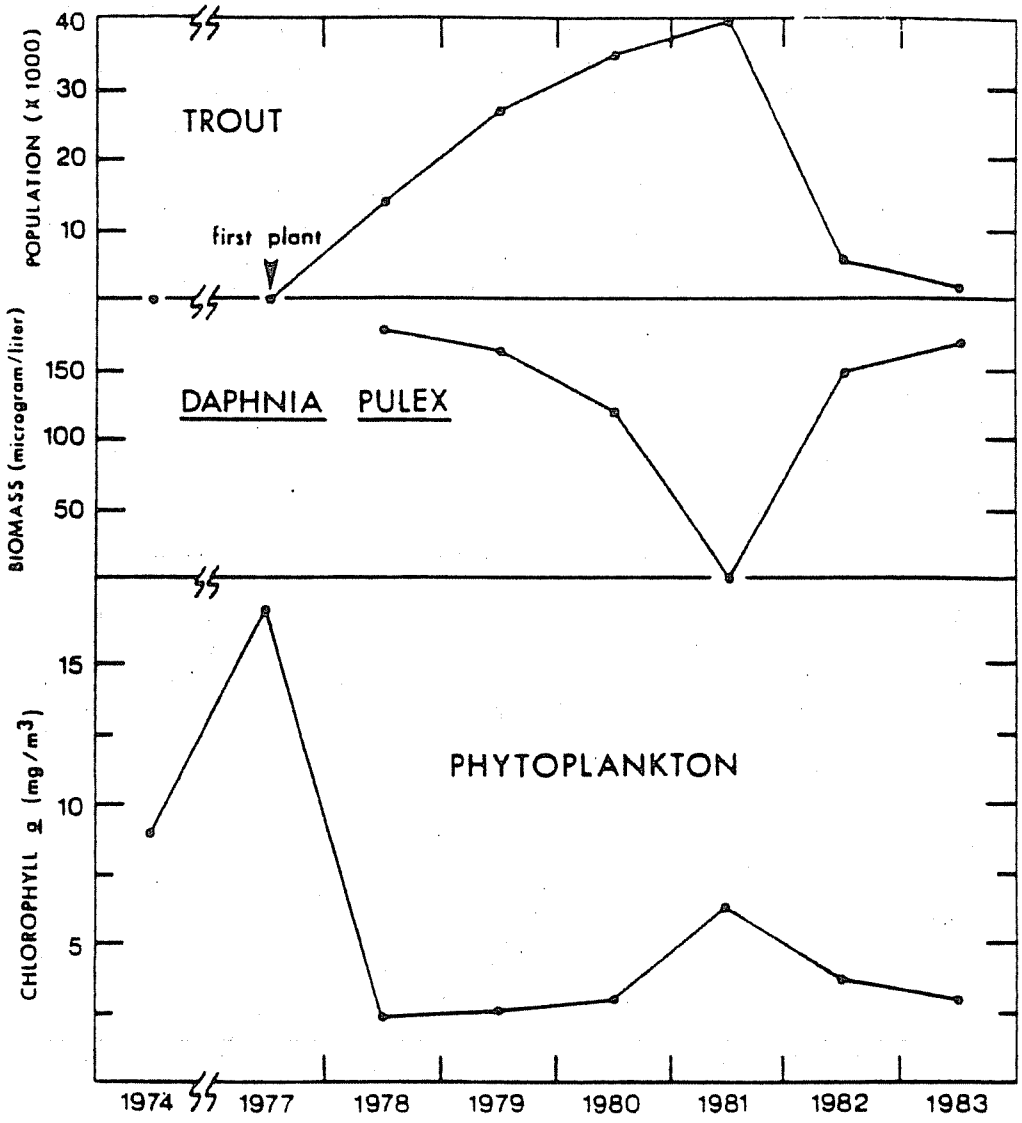

Figure 14 Effects of rainbow trout stocking on Daphnia and phytoplankton (as chlorophyll a) in previously unstocked Medical Lake, Washington. Source: Scholz et. al. 1985.

exclusively on the large *Daphnia* population. From 1978 to 1981, the average size of *D. pulex* decreased from 2.3 to 1.0 mm due to size selective predation by all age-classes of trout. Figure 14 displays the changes in standing crop of *D. pulex* and phytoplankton in relation to the rainbow trout population. *Daphnia* abundance was clearly related to the numbers of trout in the lake, steadily decreasing through 1981. Consequently, algae levels rose. These changes were almost certainly due to the reduction in grazing since phosphorus levels were relatively constant during this period.

Both the enclosure experiments and the studies on Medical Lake compared algae levels in a fishless environment with algae levels after fish stocking. Collectively, these studies suggest that a lake stocked with trout or any other plantivorous fish will contain more algae than the same lake without fish.

The situation is somewhat different in most rotenoned Washington lakes, which have been routinely stocked for many years with a fairly uniform number of trout. The lakes are fishless for only about 5-8 months following rotenone, at which time fingerling trout are restocked, generally at the prerotenone levels. Although it is logical to assume that algal abundance would return to the pre-rotenone level in such a case, no studies have actually addressed this question. Shapiro and Smeltzer (1982) did examine a somewhat analogous situation on Clear Lake, Minnesota: the lake was poisoned with toxaphene and responded with slightly increased transparency. The year after treatment, Clear Lake was restocked with both planktivorous and bottom feeders and clarity was reduced to about pretreatment levels (Figure 15).

Figure 16 shows that virtually all zooplankton and residual trout are killed by the rotenone treatment. Shortly thereafter, an algae bloom occurs owing to some combination of decreased grazing and increased phosphorus levels. This bloom subsides in the winter. Zooplankters then recover to at least their former levels of abundance; grazing itself increases due to the large, more efficient grazers that dominate while fish are absent. As a consequence, algal abundance decreases to some level that is lower than before (i.e., when fish are restocked in the lake at prerotenone levels). They crop back or even eliminate the large grazers that developed in their absence, and the zooplankton community returns in number and kind to the prerotenone state. As a consequence, algae levels increase to their prerotenone state, negating any short-term water quality benefits that might have occurred while fish were absent. (Since the fish-free period generally runs from late fall through early spring - a time of normally low algae and grazer levels - actual decreases in algal abundance may be unnoticeable, if they occur at all).

In Figure 16 is was assumed that the only fish in the lake were stocked trout, although this is not the case; both target and non-target species share the lake with trout and are killed along with

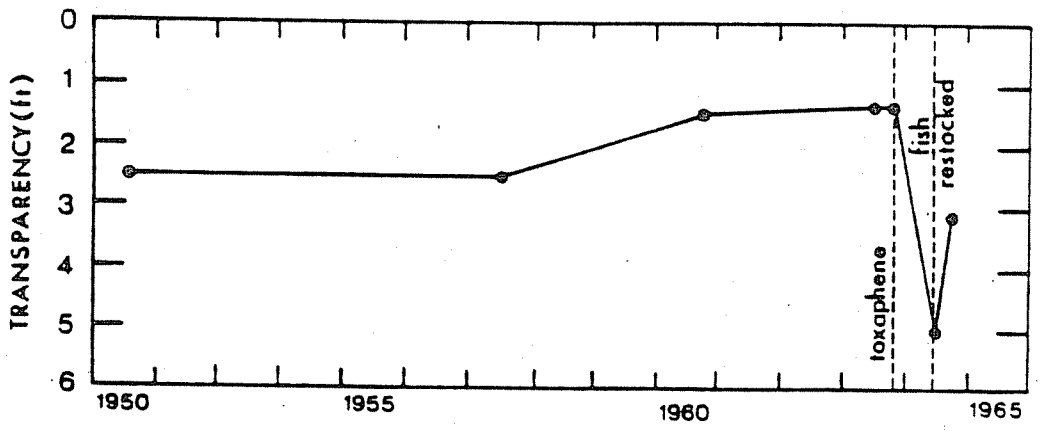

Figure 15 Effect of toxaphene poisoning and subsequent restocking with planktivores and bottom feeders on water clarity in Clear Lake, Minnesota. Source: Shapiro and Smeltzer 1982.

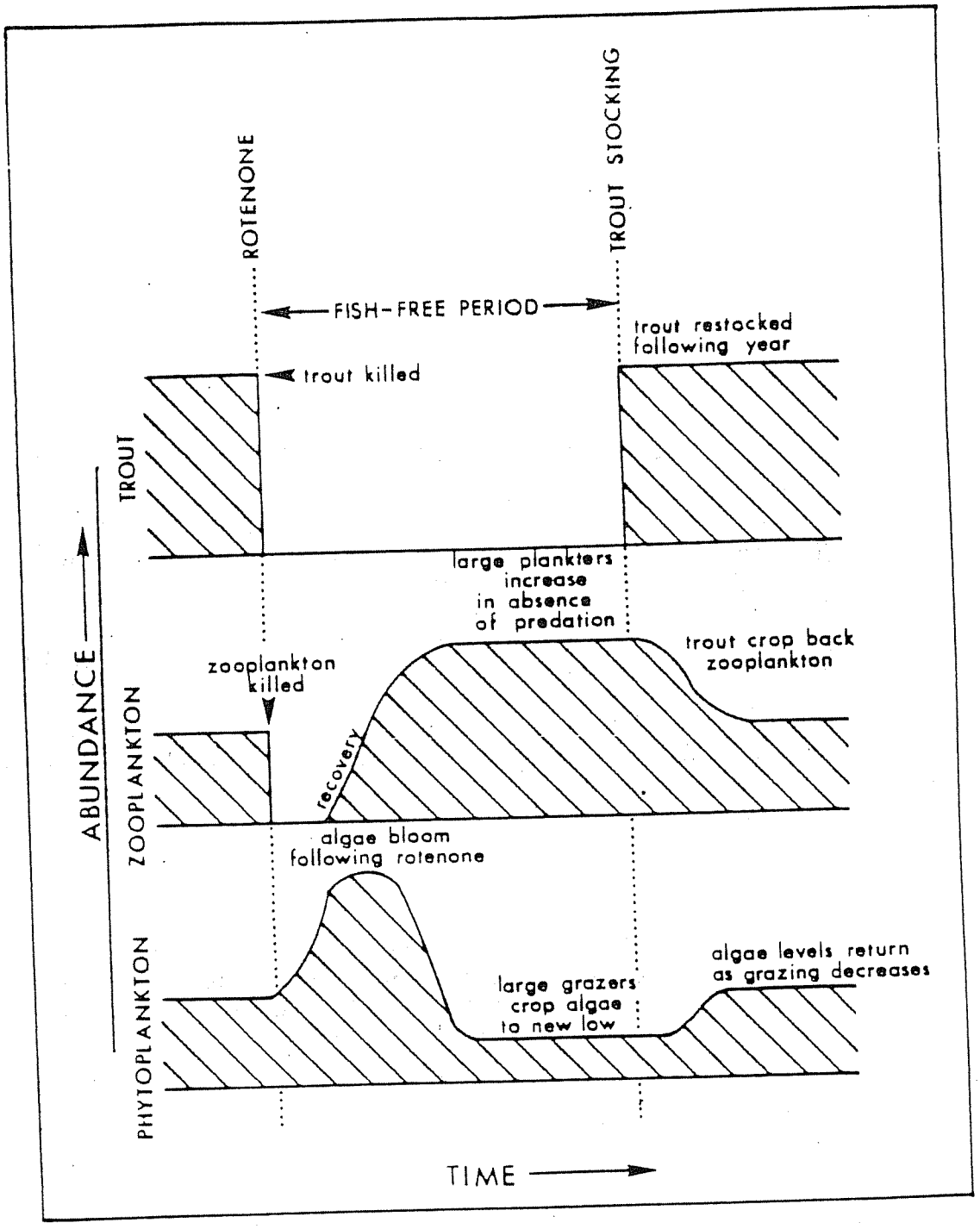

Figure 16. Hypothetical scenario following rotenone poisoning and trout restocking in a lake. Time and abundance not necessarily to scale.

them by rotenone. Many of these other fish are planktivorous, including yellow perch and sunfish which selectively feed on large grazers in much the same way as trout. Unlike the trout, which are stocked again the following spring, these other species must either repopulate the lake from survivors or be illegally reintroduced. In either case they do not usually reach their prerotenone levels of abundance for several years. If these target fish are highly planktivorous, it can be hypothesized that predation on the grazers will be somewhat reduced during this period, with trout the only important planktivores. If this hypothesis is correct, the net effect would be reduced algae levels when compared to the prerotenone years, during which time the total planktivore population was higher. There is no evidence in the literature to either support or refute this hypothesis. It is equally possible that trout alone could decimate the large grazers, even in the absence of other planktivorus fish.

Zooplankton

Short Term Effects - Table G displays the results of bioassays performed on various zooplankters. The cladocerans ("water fleas"), especially *Daphnia*, are well represented in the tests. While the 48-hour LC50's for cladocerans range widely from 0.01 ppm to 0.57 ppm, most are within the range 0.1-0.5 ppm of formulation. LC50's for the copepod *Cyclops* are somewhat lower, between 0.10 and 0.22 ppm. Based on laboratory findings, at least 50% of the cladocerans and copepods could be expected to die from exposure to the rotenone concentrations commonly used in fisheries work (0.5 ppm and up).

The effects of rotenone on the two other major components of the zooplankton community, rotifers and protozoans, have not been studied in the laboratory.

There is almost unanimous agreement among researchers that rotenone's immediate effect on the zooplankton is catastrophic. Table H shows the results of nineteen studies in lakes and ponds where zooplankton abundance was recorded before and shortly after (within four days in 12 of the studies) treatment. In 17 of the 19 cases, the immediate reduction in total numbers of mid-water zooplankton was between 75 and 100%. In 16 cases, the reduction was between 95 and 100%.

In the other two cases there was no reduction at all due to rotenone; in one of these (Libey and Holland, 1980), the very light dosage (0.1 ppm Noxfish) probably accounts for the absence of a zooplankton kill. This concurs with the bulk of laboratory bioassays cited above, which show that cladocerans and copepods (the important plankters in Libey and Holland's ponds) generally require more than 0.1 ppm to kill 50% of the population in 48

Table G Toxicity of rotenone to zooplankton in laboratory bioassays.

Species	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	References
CLADOCERA (water fleas)							
Daphnia pulex	0.01	48 hr.	16°	35 mg/l TDS		LC50	U.S. Federal Water Pollution Administration 1968
Daphnia pulex	0.025	3 hr.			5% rotenone	100% mortality	Hamilton 1941
Daphnia pulex	0.0275	24 hr.	16±1°	20 mg/l total hardness pH 6.6	Noxfish	LC50	Chandler & Marking 1982
Daphnia	0.48	48 hr.	27°		cubé	LC50	Wright 1957
	0.24	48 hr.	27°		Noxfish	LC50	
	0.32	48 hr.	27°		Pro-Noxfish	LC50	
	0.57	48 hr.	24°		cubé	LC50	
	0.49	48 hr.	24°		Noxfish	LC50	
	0.44	48 hr.	24°		Pro-Noxfish	LC50	
	0.55	48 hr.	20°		cubé	LC50	
	0.56	48 hr.	20°		Noxfish	LC50	
	0.57	48 hr.	20°		Pro-Noxfish	LC50	
Daphnia	0.1	48 hr.	27-29°	pH 7.2	4.9% rotenone	Minimum lethal dose-weakest concentration of chemical which produced a kill exceeding 25%	Zischkale 1952
Daphnia	0.100	48 hr.	16°	pH 7.4-7.8		LC50	Sanders & Cope 1966
Daphnia	0.55	48 hr.	20°		Pro-Noxfish	LC50	Brooks 1961
	0.44	48 hr.	24°		Pro-Noxfish	LC50	
	0.31	48 hr.	27°		Pro-Noxfish	LC50	
Daphnia	0.25	1 hr.				LC50	Negherbon 1959

Table 4 Continued

Species	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
Simocephalus serrulatus	0.190	48 hr.	16°			LC50	Sanders & Cope 1966
Leptodora kindtii	0.025	3 hr.			5% rotenone	100% mortality	Hamilton 1961
COPEPODA (copepods)							
Cyclops	0.22	48 hr.	27°		cubé	LC50	Wright 1957
	0.12	48 hr.	27°		Noxfish	LC50	
	0.14	48 hr.	27°		Pro-Noxfish	LC50	
	0.24	48 hr.	20°		cubé	LC50	
	0.14	48 hr.	20°		Noxfish	LC50	
	0.19	48 hr.	20°		Pro-Noxfish	LC50	
Cyclops	0.18	48 hr.	20-27°		Pro-Noxfish	LC50	Brooks 1961
	0.14	48 hr.	20-27°		Pro-Noxfish	LC50	
Cyclops	0.1	3 days	11±1°	pH 7.9 260 mg/l hardness	5% rotenone liquid	none survived	Meadows 1973
Diaptomus sicilioides	0.025	3 hr.			5% rotenone	100% mortality	Hamilton 1941

Table H Data summary of zooplankton studies in lakes and ponds. I: immediate effects of rotenone on mid-water zooplankton.

Name, location	Surface area (acres)	Formulation	Dosage (ppm)	Post-rotenone sampling	Immediate reduction in zooplankton abundance	Reference
Fern Lake western Washington	21	powdered rotenone (5% rotenone)	0.5	within 3 days	nearly 100%	Kiser et al. 1963
Celestine Lake Alberta, Canada	96	derris root powder (5% rotenone)	0.75	within 24 hours	more than 95%	Anderson 1970
Patricia Lake Alberta, Canada	170	derris root powder (5% rotenone)	0.75	within 24 hours	95-100%	Anderson 1970
McCormick Lake Nova Scotia, Canada	6	derris (5% rotenone)	1.33	within 24 hours	nearly 100%	Smith 1940
Ljøgodtjern Norway	6	Pro-Noxfish	0.5	28 days after	99%	Hongve 1977
Smith Lake Colorado	24	derris powder (5% rotenone)	1.0	within 4 days	nearly 100%	Hoffman & Olive 1961
Velká Arazimova Czechoslovakia	0.1	"Lonchocarpus extract"	---	7 days after	nearly 100%	Hrbáček & Novotná-Dvořáková 1965
West Pond Montana	13	Pro-Noxfish	0.7	within 1 month	0%	Wollitz 1962
Third Sister Lake Michigan	10	derris root powder (5% rotenone)	0.5	within 24 hours	100%	Brown & Ball 1943a
South Branch Lake (cove only) Maine	11	Noxfish	0.6	within 24 hours	97%	Neves 1975
Middle Pond Montana	20	Chem-Fish Special	0.7	within 1 month	98%	Wollitz 1962

Table H Continued

Name, location	Surface area (acres)	Formulation	Dosage (ppm)	Post-rotenone sampling	Immediate reduction in zooplankton abundance	Reference
Silver Lake western Washington	106	powdered rotenone (5% rotenone)	1.0	2 days later	100%	Kiser et al. 1963
Halmsjön Sweden	94	Pro-Noxfish	0.5	3 days after 10 days after	96% 97%	Almquist 1959
Potter's Lake New Brunswick, Canada	113	derris powder (5% rotenone)	0.5	"after"	nearly 100%	Smith 1941
Ponds A & B Indiana	1	Noxfish	0.1	within 3 days	0%	Libey & Holland 1980
Bug Lake Wisconsin	11	Pro-Noxfish	2.5	within 12 days	100%	Serns 1979
Carls Lake Minnesota	110	Chem-Fish Pro-Noxfish	3.0 3.0	within 12 days	100%	Bandow 1980
Wirth Lake Minnesota	40	"rotenone"	---	within 10 days	99%	Shapiro 1982
Deming Lake Minnesota	13	derris powder (5% rotenone)	0.5	4 days later	75%	Hooper 1948

hours. Libey and Holland's research is interesting in that it is one of the few reported cases where rotenone was used to purposely poison zooplankton; the authors wished to starve a stunted bluegill population by reducing the plankters.

The results of the other study in which there was no zooplankton kill (Wollitz, 1962 on West Pond, Montana) are difficult to explain, especially since the same author noted a 98% reduction in the similarly treated nearby Middle Pond (Wollitz, 1962).

Zooplankters feel the effects of rotenone shortly after application: Kiser et al. (1963) made tows every 15 minutes on Silver Lake, Washington, during the day it was treated, noting a 34% decrease in plankton counts within 30 minutes after treatment began. The greatest reduction in total zooplankton counts came between 15 minutes and one hour after treatment began, a reduction of 70%.

Susceptibility of Different Species - While virtually all plankters are affected by rotenone, some are more tolerant than others.

There is general agreement that the planktonic crustaceans, especially the cladocerans, are the group most quickly or thoroughly eliminated (Anderson, 1970; Bandow, 1980; Hrba'cek and Novotna'-Dvora'kova', 1965; Hongve, 1977; Neves, 1975; Smith, 1940; 1941; Wollitz, 1962; Brown and Ball, 1943a; Almquist, 1959; and Hooper, 1948). Almquist (1959) ranked various plankters according to their sensitivity to rotenone in Lake Erken, Sweden, and found that among the ten most sensitive, eight were cladocerans and two were rotifers. *Diaphanosoma* and *Daphnia* required the lowest exposure (all died between 30 minutes and two hours in 0.5 ppm formulation) of all test animals. Almquist found wide differences in tolerance even among the cladocerans, however; *Alonella* and *Pleuroxus* withstood 1.5 ppm and 2 ppm Pro-Noxfish for up to 8 hours, and *Alona* was one of the most tolerant of all 44 organisms test, requiring seven hours in 4.5 ppm Pro-Noxfish for a 100% kill. Kiser et al. (1963) reported these same three genera resisting rotenone in Fern Lake, Washington, though habitat within the lake may have contributed. And *Bosmina* remained present in the open water of Silver Lake, Washington after treatment far longer than *Daphnia* or *Holopedium* (Kiser et al., 1963).

Rotifers are generally considered to be more tolerant of rotenone than the cladocerans or copepods. *Keratella* has been singled out as highly resistant by several authors (Bandow, 1980; Almquist, 1959; Anderson, 1970; Smith, 1940; 1941; Walters and Vincent, 1973; Neves, 1975), along with *Conochilus* (Neves, 1975; Smith, 1941; Almquist, 1959).

Susceptibility According to Habitat - Sensitivity to rotenone apparently varies not only to the species of plankter, but with the habitat type within the lake as well, though only one study has

adequately addressed this question. Kiser et al. (1963) separately sampled three different habitats in Fern Lake, Washington: the open water, the margin between the brush and open water, and the shallow weedy shoreline. Immediately after treatment they noted that the reduction in total zooplankton counts was most severe in open water and least severe in the weedy shoreline. The margin was intermediately affected.

Naturally, as each habitat supports a different assortment of plankters, it could be concluded that these results were mostly due to the varying sensitivities of the species involved rather than the habitat. But a few plankters in Fern Lake, such as the cladoceran *Alonella* and the rotifer *Chydorus sphaericus*, live in all three habitats, and they suffered greater losses in the open water than in the weedier areas.

It is well documented that vegetation and heavy organic debris detoxify rotenone, and the Fern Lake researchers suggested this as a reason why the weedy area plankters were somewhat less affected. They also admitted the possibility that the inaccessible brushy regions weren't as well dusted with rotenone. All other field studies have confined themselves to open-water sampling, or failed to break down their results by habitat type.

Long Term Effects - Recovery - Although they are drastically reduced immediately following rotenone treatment, zooplankton communities do recover in almost all cases. Even in those lakes where not a single living plankter appeared in the post-rotenone samples, enough escaped or survived treatment to eventually repopulate the lake.

As previously noted, some plankters escape treatment in densely weeded areas where rotenone is quickly detoxified (Almquist, 1959; Kiser et al., 1963). Others may survive simply by virtue of their tolerance to rotenone. Certain plankters may survive by means of parthenogenetic summer eggs and tough ephippial eggs which are unaffected by rotenone (Bandow, 1980; Anderson, 1970; Kiser et al., 1963). Both cladocerans and cyclopoid copepods produce ephippial eggs, which lie dormant in the lake sediments throughout the winter. They are normally produced in the late fall, but unfavorable environmental factors may stimulate early production. Kiser et al. (1963) observed female cladocerans with early ephippial eggs, and suggested that it was the rotenone that acted as this unfavorable factor. Finally, it has been suggested that zooplankton for repopulation may also come from other nearby bodies of water (Hrba'cek et al., 1961; Kiser et al, 1963), though this has never been documented in rotenoned lakes.

In most lakes there is a period following the rotenone treatment during which plankters (at least crustaceans) are scarce or absent from tow samples. Table I shows the results of 15 studies where the long-term effects of rotenone on zooplankton were recorded. In

Table I Data summary of zooplankton studies in lakes and ponds. II: long-term effects of rotenone.

Lake	Rotenone formulation	Dosage (ppm)	Time that Lake was toxic to fish	Crustacean-free period	Rotifer-free period	Fish restocked or re-entered	Period of study before rotenone	Period of study after rotenone	Time to complete recovery	Any species fail to reappear?	Any new species appear?	Reference
Fern Lake western Washington	powdered rotenone (5% rotenone)	0.5	33 days	2 weeks	---	steelhead	2 years	6 months	17 weeks	no	yes	Kiser et al. 1963 Fowler 1973
Celestine Lake Alberta, Canada	derris root powder (5% rotenone)	0.75	---	9 months	never absent	rainbow trout	1 month	2 years, 1 month	3 years	yes	yes	Anderson 1970
Patricia Lake Alberta, Canada	derris root powder (5% rotenone)	0.75	---	6 months	never absent	rainbow trout	1 month	3 years, 5 months	3 years	no	yes	Anderson 1970
McCormick Lake Nova Scotia, Canada	derris (5% rotenone	1.33	~1 month	9 months	never absent	---	3 days	1 year, 3 months	1 year	no	yes	Smith 1940
Ljøgoattjern Norway	Pro-Noxfish	0.5	---	---	---	rainbow trout	1 year, 3 months	1 year, 2 months	1 year	no	yes	Hongve 1977
Smith Lake Colorado	derris powder (5% rotenone	1.0	---	3 months	never absent	---	14 days	11 months	6 months			Hoffman & Olive 1961
Velká Arazimova Czechoslovakia	"Lonchocarpus extract"	---	---	1 month	never absent	carp, bass, roach, bream	2 years	4 years, 1 month	4 months	yes	yes	Hrbáček & Novotná-Dvořáková 1965

Table I Continued

Lake	Rotenone formulation	Dosage (ppm)	Time that lake was toxic to fish	Crustacean-free period	Rotifer-free period	Fish restocked or re-entered	Period of study before rotenone	Period of study after rotenone	Time to complete recovery	Any species fail to reappear?	Any new species appear?	Reference
West Pond Montana	Pro-Noxfish	0.7	less than 17 days	---	---	---	1 year, 3 months	9 months	immediate	---	---	Wollitz 1962
Third Sister Lake Michigan	derris root powder (5% rotenone)	0.5	7 days	never absent	never absent	none	1 year	1 year	2 months	yes	---	Brown & Ball 1943a
South Branch Lake (cove only) Maine	Noxfish	0.6	---	never absent	never absent	---	2 weeks	2 weeks	1 week	no	---	Neves 1975
Emmaline Lake Colorado	Pro-Noxfish	1.0	---	---	never absent	cutthroat trout	2 months, 2 weeks	4 years	2 years	---	---	Wrenn 1965 Walters & Vincent 1973
Potter's Lake New Brunswick Canada	derris powder (5% rotenone)	0.5	between 18-45 days	2 months	never absent	brook trout	52 days	11 months, 9 days	10 months	---	---	Smith 1941
Ponds A & B Indiana	Noxfish	0.1	---	---	---	---	1 year, 2 months	1 month, 2 weeks	immediate	---	---	Libby & Holland 1980
Bug Lake Wisconsin	Pro-Noxfish	2.5	5 months, 20 days	5 months, 19 days	---	fathead minnows, brook trout	3 months, 24 days	2 years	7-8 months	yes	yes	Serns 1979
Car's Lake Minnesota	Chem-Fish Pro-Noxfish	3.0 3.0	---	26 days	never absent	bass, walleye, channel catfish, brown trout	1 year, 3 months	2 years	did not fully recover	no	---	Bandow 1980

11 of these investigations, there were sufficient data to establish the length of time following treatment that plankters were absent from open-water tows.

In two examples, some crustacean plankton was always present in tow samples, but these appear to be special cases: Neves (1975) poisoned only an isolated cove within a lake, and immigration from the non-treated areas took place immediately following treatment; and Brown and Ball (1943a) observed an unusually short toxic period of seven days, possibly accounting for the continued presence of plankters in small numbers.

In the other nine cases, cladocerans and copepods were entirely absent from open-water tow samples from two weeks to as long as nine weeks.

Considering first the four lakes in which crustacean plankters remained absent for the longest time (Serns, 1979; Anderson, 1970; Smith, 1940):

Serns found no crustacean plankters for five months and nineteen days following treatment in Bug Lake, Wisconsin. The lake was toxic to fish, however, for at least five months and twenty days, possibly as a result of the heavy dosage (2.5 ppm Pro-Noxfish).

It is somewhat more difficult to understand the other three lakes where the crustacean-free period lasted from six to nine months (Smith, 1940; Anderson, 1970). While dosages were somewhat higher (0.75 ppm-1.33 ppm) than the bulk of the lakes studied, Bandow (1980) and Hoffman and Olive (1961) saw crustaceans much sooner after using 3 ppm and 1 ppm. Sampling bias may be partly responsible in the example of McCormick Lake (Smith, 1940); tows were made for only two months following treatment with rotenone, after which there was a seven-month period when no samples were taken. In the case Anderson's (1970) two lakes, the extremely long absence of crustacean plankton (6-9 months) may well be due to the oligotrophic nature of the high mountain lakes involved. Anderson (1972) and Wrenn (1965) pointed out that plankton recovery was slower in the relatively sterile alpine lakes than in nutrient-rich lowland lakes.

For Washington's rehabilitated lakes, the best estimate of this crustacean-free period probably lies between two and twelve weeks, the range of the other five studies (Kiser et al., 1963; Hrba'cek and Novotna'-Dvora'kova', 1965; Bandow, 1980; Smith, 1941; and Hoffman and Olive, 1961).

Since cladocerans and copepods are the plankters that juvenile trout eat most frequently, this period when they are virtually absent from open water may have important management implications in cases where restocking is planned shortly after treatment.

Generally, this is not the case in Washington; 78% of the lakes in the program have been treated in the fall and restocked no sooner than five months later. On several cases crustacean plankton reappeared before the lakes were nontoxic to fish.

Factors Affecting Recovery of Different Plankters - Once plankters reappear, the community begins to rebuild itself, eventually returning in most all cases to prerotenone levels of abundance and diversity. But just as the various plankters respond differently to rotenone when it is applied, they also recover at different rates.

Anderson (1970) stated that the speed of recovery for different plankters was likely related to four factors:

1) Susceptibility to rotenone . Most researchers found that the plankton groups most tolerant of rotenone recovered the quickest. Rotifers usually reached prerotenone levels of abundance before the cladocerans and copepods. In Smith Lake, Colorado, rotifers recovered after five months, while the crustacean plankters required six months (Hoffman and Olive, 1961). Hrba'cek and Novotna'-Dvora'kova' (1965) found cladocerans and copepods recovered between 3 and 4 months after poisoning, while protozoans and rotifers reappeared in just 1 - 2 months. In the alpine lakes studied by Anderson (1970), the rotifers had completely recovered to their former levels of diversity and abundance in 11 - 12 months, a full two years before the crustaceans did so.

2) Time of reproduction . Anderson (1970) states that rotenone is more devastating to those species which have not reached reproductive maturity by the time rotenone is applied. This was the case with the copepod *Diaptomus sicilus* in a Canadian alpine lake, and it was the last species to recover. In general, the major reproductive peak occurs in the spring, with a lesser one in the fall; but the precise timing depends on the species and water conditions (Arni Litt, UW, pers. comm.).

3) Ability to form resistant stages . All cladocerans, copepods, and rotifers have the ability to form ephippial or other over-wintering eggs; in Washington this occurs mostly in the eastern half of the state where ice cover forms (Arni Litt, UW, pers. comm.). Anderson (1970) suggests that such eggs - depending on when in the fall a particular species produces them - could resist the poisoning and aid in recovery. Bandow (1980) suspected that this ability allowed *Daphnia* to become the dominant crustacean in a rotenoned lake. Brynildson and Kempinger (1973) stated that the "comeback" of *Daphnia* in a Wisconsin lake after rotenone may have been partly due to ephippia which hatched the following spring.

Partial Recovery - Long before zooplankton communities recover to the point where all species have reappeared in their approximate prerotenone levels, there is usually a point when most species are present in large numbers.

Anderson (1970) noted that even in the extreme case of alpine lakes where complete recovery took as long as three years, most species of crustaceans had reappeared within 10 months of poisoning. More relevant to Washington's lowland lakes is the case of Fern Lake, Washington, where although complete recovery in all habitat types took 17 weeks, the authors suggested that zooplankton populations had recovered to the point where trout could be restocked in just 9 - 10 weeks (Kiser et al., 1963). Two weeks later, all open water crustaceans had returned to prerotenone levels. WDW restocked Fern Lake 37 days after poisoning, and the authors suggested that this may have been about five weeks too soon in view of the reduced plankton levels.

During this period to complete recovery, there are often shifts in the zooplankton community structure. One of the more gross changes is the temporary disappearance of the cladocerans and copepods, while rotifers dominate. Researchers have reported other unusual changes in the community during recovery as well: Neves (1975) noted minor rotifer "blooms" during recolonization of a poisoned lake cove, probably due to lake of competition and/or low predation by grazing plankters. Walters and Vincent (1973) also observed a temporary rotifer "bloom" during recovery. Patricia and Celestine lakes saw an increase in small sized cladocerans (Anderson, 1970). In Fern Lake, a number of weedy shoreline plankters unaffected by rotenone invaded the open-water areas of the lake and became dominant for about nine weeks. By the twelfth week they had been gradually excluded by the original open-water species which had returned. (Kiser et al., 1963). Anderson (1970), though he did not sample the shoreline areas, noted "new" species in the open-water tows on Patricia and Celestine lakes, and suggested that the same invasion by resistant shoreline plankters seen in Fern Lake was occurring. Neves (1975) did not observe this phenomenon in a treated cove, claiming that quick recolonization from outside by open-water plankters was the reason.

These changes in community structure were all relatively minor and temporary. Probably the most commonly observed change in zooplankton community structure during the recovery period is the dominance of large sized cladocerans after treatment (Bandow, 1980; Hrba'cek and Novotna'-Dvora'kova', 1965; Anderson 1970; Gustafson et al., 1981; Serns, 1979; Walters and Vincent, 1973; Stenson, 1972). All of these authors trace this change to the absence of predatory fish in the lake following poisoning. And, depending on whether or not fish are restocked, the change can be temporary or permanent.

Complete Recovery - Zooplankton recovery times in 15 test waters are shown in Table I. Figure 17 graphically displays the recovery time on most of these waters. For interpretation of these results "complete recovery time" means the time it took for all, or nearly all, of the important or sampled elements of the zooplankton community to reappear and reach approximate prerotenone levels of abundance. In a number of instances, the authours have actually stated a recovery time. In others, recovery time must be inferred from the data. In both cases, there are three main reasons why the assignment of recovery times must be regarded as an approximation. 1) the zooplankton comunity cannot be expected to reappear exactly as before rotenone, there is often a characteristic shift to larger sized plankters in the absence of predatory fish; 2) some studies identified samples down to the generic or specific level, while others used only broad taxonomic descriptions such as "rotifers" and protozoans". This makes it difficult to establish when diversity has been restored; and 3) zooplankton counts vary widley from year to year, making it difficult to establish prerotenone levels of abundance to use as a "yardstick" in measuring recovery.

The most reliable studies are those in which plankters were identified to the generic or specific level, and samples were collected regularly for several years prior to treatment. Table I shows that recovery times ranged from "immediate" to three years, and in one case (Bandow, 1980) recovery was not complete when the study ended two years after rotenone treatment.

Carls Lake, Minnesota (Bandow, 1980) appears to be a special case: while calanoid copepods never returned to full abundance even after two years, there was a sharp increase in total standing crop of zooplankton, mostly *Daphnia*. Several other factors confuse the picture on Carls Lake: the severe winterkill that disrupted the ecosystem not long before treatment, the double application of rotenone at high dosage (3 ppm), and the post-rotenone introduction of at least six species of fish. The combined effect of these unusual variables makes it hard to draw conclusions on recovery time from this study. Other special cases include Libey and Holland (1980), where the small dosage never affected zooplankton; Wollitz's (1962) treatment of West Pond, an anomaly; and Neves (1975), where the poisoned cove recovered completely in one week due to rapid immigration from the untreated areas.

The remaining examples all show recovery times ranging from two months to three years. Where complete recovery required two and three years (Emmaline, Celestine and Patricia Lakes), it is perhaps relevant to note that all three are oligotrophic alpine lakes. Zooplankton in sterile alpine lakes require an unusually long time to recover (Anderson, 1972; Wrenn, 1965). Moreover, Anderson and Wrenn were only able to sample the lakes one and two months prior to poisoning, making it difficult to say with certainty what the prerotenone abundance levels were.

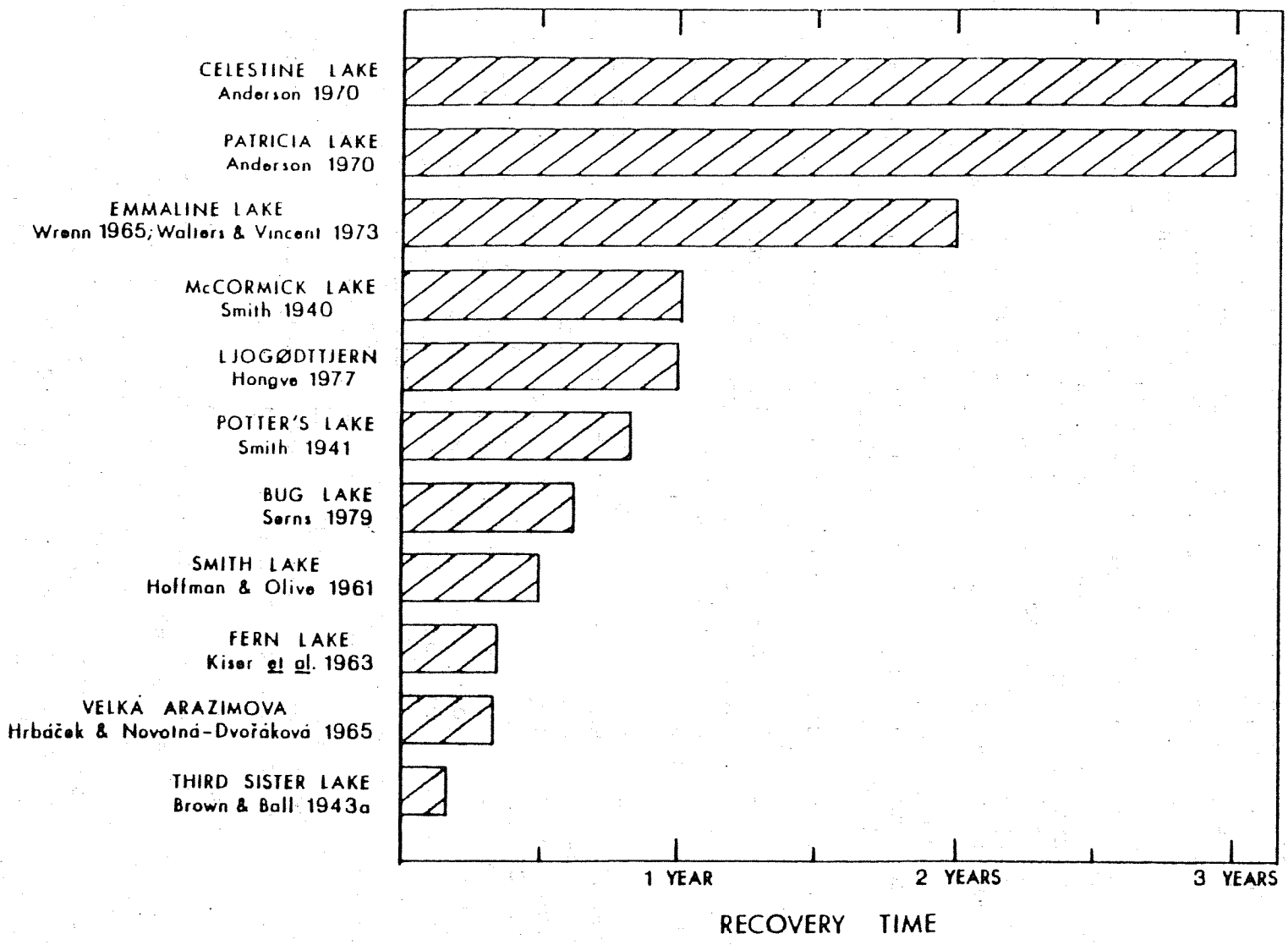

Figure 17. Recovery times for the zooplankton in several lakes and ponds following rotenone. Data are from Table 17.

Complete recovery based on the remaining studies required anywhere from two to twelve months. Eliminating the two month example (Brown and Ball, 1943a) due to their unusually short toxic period for fish, we are left with a range of four months to a year. The most thorough of these studies (based on the three criteria discussed above) is that of Kiser et al. (1963) on Fern Lake, Washington, where recovery took 17 weeks.

In several lakes cited in Table I, zooplankton populations not only recovered to previous levels after poisoning, but exceeded them (Hongve, 1977; Anderson, 1970; Woolitz, 1962; Serns, 1979).

Since none of these four studies involved extensive sampling before treatment, the "increased" populations may not be significant.

The dosages used in most of the studies cited are somehwat less than the statewide average applied in Washington (1.23 ppm). Considering the effect of different lake chemistries on rotenone as well as other variables, it is impossible to say whether or not this is significant. There is no apparent correlation between dosages shown in Table I and the corresponding times to complete recovery. There is also no clear correlation between recovery and either pH or water temperature, despite the fact that a wide range of pH's (5.9-8.9) and water temperatures (38° - 82°F) are represented in the test waters. Two other authors have made statements on recovery time that should be mentioned here, although their data cannot be included in Table I: Schnick (1974), after a review of the literature, concluded that "recovery takes from 1.5 - 3 months"; possibly referring to what has been described as "partial" recovery, where most but not all the important elements of the plankton community have reappeared. Galbraith (1974), reviewing unspecified data on Michigan trout lakes, stated that after rotenone "it takes at least on full year before the *Daphnia spp.* regain their original densities."

Brynilsdon and Kempinger (1973) recommended speeding up the recovery of *Daphnia, Leptodora,* and *Holopedium* in rotenoned Wisconsin lakes by stocking these crustacean plankters shortly after treatment.

Disappearance of Species/Appearance of New Species - Table I shows that in four of ten cases, a species observed before treatment failed to reappear in samples taken after recovery was "complete". In three of these cases (Anderson, 1970; Brown and Ball, 1943a, Serns 1979), the authors suggested that incomplete sampling or the sporadic prerotenone appearance of a rare specimen was responsible for the "disappearance". In the other example, the disappearance of *Daphnia cucullata* from a pond was traced to exclusion by a larger daphnid in the absence of fish (Hrba'cek and Novotna'-Dvora'kova', 1965).

None of the 42 crustacean species in Fern Lake, Washington, disappeared permanently after rotenone (Kiser et al.,1963), and the authors believed that complete elimination of a species was "quite unlikely".

The post-rotenone appearance of species never collected before treatment was common, although not explained or assigned any significance. Kiser et al. (1963) observed that after treatment, many weedy-shoreline plankters invaded open-water areas where they were normally never found. Had the authors not towed the shoreline, these species may have been classed as "new" to the lake. Undoubtedly this happened in some of the other lakes, where shoreline habitat was not sampled. And, since only two of the other studies where "new" species were reported included more than a few months sampling before treatment (Hongve, 1977; Hrba'cek-Novotna'-Dvora'kova', 1965), the chances of missing a seasonal or sporadic species were very high in the others.

These "new" species appearing after rotenone never attained dominance in any of the lakes cited in Table I. Even in the case of Fern Lake, Washington, the large population of cladocerans which dominated nearby untreated lakes never gained a foothold in Fern Lake during its recovery (Kiser et al., 1963).

Fish/Zooplankton Interactions - Because fish are consumers near the top of a lake's trophic "pyramid", and because they make up only a small percentage of the lake ecosystem's total matter and energy, they were once considered unimportant in controlling the plankters. There is now a great deal of evidence to the contrary; fish can and do have a dramatic influence on the zooplankton in a lake (Shapiro et al., 1975; Brooks and Dodson, 1965; Galbraith, 1967).

While zooplankton is not always the main food source for fish (Walters and Vincent, 1973), almost all fish in a lake eat zooplankton to some degree, at some life stage. Naturally, a great deal depends on the lake itself and what other foods it supplies. In general, though, rainbow trout of all ages and sizes often feed heavily on zooplankton in Washington lakes, mainly cladocerans and copepods (Wydoski and Whitney, 1979; Carlander, 1969). They do not feed indiscriminately; instead, they individually select and eat only the largest cladocerans (Galbraith, 1967).

It is not only trout that feed on zooplankton; many of the nonindigenous fish that are targeted for eradication from Washington's trout-only lakes also eat zooplankton. Yellow perch (*Perca falvescens*) of all ages and sizes prey on cladocerans and copepods (Wydoski and Whitney, 1979; Serns and Hoff, 1984) and have been called one of the most important zooplanktivores in the U.S. (Shapiro et al., 1975). Like trout, they select and eat only the largest plankters (Galbraith, 1967; Serns and Hoff, 1984). Zooplankton is important in the diet of fathead minnows (*Pimephales promelas*) (Serns, 1979; Galbraith, 1974), pumpkinseed sunfish

(*Lepomus gibbosus*) (Beard, 1971) and bluegills (*Lepomis marcochirus*) (Krska and Applegate, 1984; Shapiro et al., 1975). Even the brown bullhead catfish (*Ictalurus nebulosus*), commonly regarded as a bottom feeder, is often planktivorous (Bandow, 1980), at times exclusively so (Olson and Koopman, 1976).

When all, or almost all the fish in a lake are eliminated, zooplankton return in large numbers within 2 - 10 weeks of treatment, usually closer to three weeks and major shifts in dominance during the period of time before fish are reintroduced to the lake may reasonably be expected.

In some lakes, when the period of time before reintroduction is very short (e.g., 37 days), nothing of importance happens (Kiser et al., 1963). But most lakes in Washington treated with rotenone in the fall or winter are restocked the following spring, allowing at least five months during which the zooplankton suffer no predation.

Many investigators have reported a shift in community structure, with some plankters exceeding prerotenone levels while others decline. By far the most common shifts observed were:

1) an increase in the number of large sized plankters (usually *Daphnia*), with a corresponding decline in the smaller-sized plankters that cannot graze as effectively; and

2) an increase in the body size of the already existing plankters.

Table J show the results on six lakes where body-size relationships were examined following rotenone and fish stocking. In all six lakes, large-sized plankters became dominant when fish were absent or scarce; and in the two studies where carapace lengths were measured, the small-sized plankters increased in body size (this may have occurred in the other four lakes as well). In all but one lake, the large-sized species that became dominant already existed in the lakes but in smaller numbers; in Lake Sarvsjon, however, the new dominant plankters had never before been recorded in the lake (Gustafson et al., 1981). In all six cases, the authors stated that these shifts were due to the absence of predation by fish following rotenone. And in every case, the zooplankton community reverted to the "normal" prerotenone conditions once fish were restocked and firmly established again.

Stenson (1972) confirmed these results with an experiment in eight Swedish lakes, which all contained the same types of fish and zooplankton. He poisoned four with rotenone and left the other four as untreated "controls". Zooplankton began to repopulate all four rotenone lakes, and Stenson stocked new fish species (trout) in three of them, while allowing the original species (perch, pike, eels) to re-enter the fourth. During the experiment, predation was low in the newly stocked "trout" lakes, but quickly returned to

Table 3 The effects of rotenone treatment and subsequent fish stocking on the kinds and size of zooplankton in six lakes.

Test water, location	BEFORE ROTENONE: Fish present before rotenone	FISH-FREE PERIOD AFTER ROTENONE: Increase in body size of zooplankton	FISH-FREE PERIOD AFTER ROTENONE: Large-sized species more abundant	FISH & ZOOPLANKTON RE-ESTABLISHED: Fish introduced after rotenone	FISH & ZOOPLANKTON RE-ESTABLISHED: Did community revert to pre-rotenone state?	Reference
Patricia Lake Alberta, Canada	lake chub mountain whitefish longnose sucker lake trout brook trout rainbow trout	not studied	yes	brook trout rainbow trout	yes	Anderson 1970
Celestine Lake Alberta, Canada	lake chub rainbow trout	not studied	yes	rainbow trout	yes	Anderson 1970
Velká Arazimova Czechoslovakia	perch roach bitterling carp	not studied	yes	carp largemouth bass roach bream	yes	Hrbáček & Novotná-Dvořáková 1965
Lake Särvsjön Sweden	whitefish perch northern pike	yes	yes	brown trout arctic char	not studied	Gustafson et al. 1981
Carls Lake Minnesota	brown bullhead bluegill brown trout channel catfish fathead minnow golden shiner green sunfish largemouth bass northern pike walleye	yes	yes	brown bullhead largemouth bass walleye channel catfish brown trout	unclear	Bendow 1980
Emmaline Lake Colorado	brook trout	not studied	yes	cutthroat trout	yes	Walters & Vincent 1973

normal in the lake repopulated by the original fish. Stenson's results are shown in Table K.

Table K. The effects of rotenone treatment and subsequent fish stocking on the kinds and size of zooplankton in eight Swedish Lakes. Source: Stenson, 1972.

Treatment	Increase in Body Size of Plankters	Shift in Dominance to Large Cladocerans
NO ROTENONE (Control)		
-Original fish species	NO	NO
-High predation (4 lakes)		
ROTENONE		
-Original fish species re-entered	NO	NO
-High predation (1 lake)		
ROTENONE		
-New fish species stocked	YES	YES
-Low predation (3 lakes)		

Clearly, the scarcity of fish in the newly stocked trout lakes allowed the larger cladocerans to become dominant, and also allowed the mean body size of the cladoceran *Bosmia* to increase. Most interesting are the results in the single rotenoned lake where the original fish repopulated after rotenone; the zooplankton community recovered and was identical to the nonrotenoned lakes. These results concur with those in Table J where communities reverted to their prerotenone state once fish were restocked. Stenson showed conclusively that it was the lack of predation, not the rotenone that changed the community.

In general, large daphnids are not found in lakes with many planktivorous fish, although there are some notable exceptions, i.e., Lake Washington (Edmondson and Litt, 1982).

There is concern that when trout are stocked following poisoning that not only will they return the zooplankton community to former levels of abundance, but that they will eventually eliminate it.

When trout are stocked in formerly fish-free lakes, for example, dominant plankters are often dramatically reduced or eliminated. Anderson (1972) stocked trout in an alpine lake that had never supported a fish population before, and with no other food source available (e.g., benthic invertbrates), they eliminated the dominant plankters within two to six years. When rainbow trout were introduced into Medical Lake, Washington, they largely eliminated the dominant plankter, *Daphnia pulex*. Knapp and Soltero (1983) felt that this loss of a preferred food item would jeopardize the newly established trout fishery.

Most of the Washington state lakes treated with rotenone have been routinely stocked with fingerling trout for many years and poisoned at more or less regular intervals. To be considered for lake rehabilitation, the lake must provide good fingerling survival and growth as indicated by yearly gill-net sets and creel checks. This empirical evidence suggests that trout stocking at historical levels does not reduce zooplankton to the point where trout growth is affected.

There is also no evidence in the literature to suggest that continued stocking in traditionally successful trout waters eliminates zooplankton as a food source. Galbraith (1967) reported that trout reduced the *Daphnia pulex* population in Sporley Lake, Michigan, to the point where the fishery deteriorated. Further research, however, showed that perch, fathead minnows, and smelt were important contributors to the *Daphnia* decline; even when trout stocking was discontinued for four years, the daphnid population stayed at very low levels. Only after the perch, fathead minnows, and smelt were poisoned with rotenone did *Daphnia* return (Galbraith, 1974).

A similar situation developed on Nebish Lake, Wisconsin, after rotenone; while both hatchery trout and yellow perch preyed heavily on large *Daphnia*, it was the exploding perch population that eventually overgrazed the lake (Brynildson and Kempinger, 1973).

The data from lakes with established fish populations at the time of rotenone treatment (Kiser et al., 1963; Anderson, 1970; Hrba'cek and Novotna'-Dvora'kova', 1965; Walters and Vincent, 1973; Stenson, 1972) show that when fish are restocked, the zooplankton community returns in kind and number to the prerotenone state.

In those lakes which contain planktivores other than trout (such as yellow perch, fathead minnows, bluegills, etc.), it is reasonable to assume that even after restocking with trout, there could be a net decrease in predation on zooplankton due to the absence of the other planktivorous fish. If this occurred, it would be a temporary situation, since the target fish populations usually re-establish themselves after a few years.

Benthic Fauna

Short Term Effects - Table L displays the results of bioassays performed on various benthic animals found in lakes and ponds. The widely cited results of Leonard (1939) have been omitted; in his tests, Leonard found that rotenone dosages as high as 2 ppm had no effect on a variety of benthic animals, but since that time, several authors have cast doubt on the quality of his rotenone formulations (Almquist, 1959; Kiser et al., 1963). Many of these studies were performed before the standarization of laboratory toxicity test (96-hour LC50's being the current standard), so it is impossible to perform any meaningful quantitative comparison which includes all the data. In addition, all of the research except for that of Zischkale (1952) involved testing of benthic animals in bare aquariums devoid of any natural substrate. Since Lindgren (1960) has shown this to be an important, if not overriding factor in benthic mortality with rotenone, the results cannot be reliably extrapolated to a real lake environment.

Laboratory tests are not without value since they can be used to understand the relative susceptibilities of different benthic animals. Figure 18 broadly groups several types of benthic animals, giving a rough idea of the varying susceptibility of each to rotenone. Data are drawn from Table L, utilizing only LC50's for exposures ranging from 24 to 96 hours. Some other data from Table L are included as well, where tests indicated that a particular concentration killed 50% of the animals; in a strict sense, these are not LC50's, though their inclusion here is justified since they provide extra data.

Figure 18 shows the decapod crustaceans (mostly crayfish) to be the most tolerant group, followed in descending order by caddisfly larvae, aquatic snails, and clams, the larval stages of dragonflies and damselflies, phantom midges, true midges and mayflies. This figure includes all the important components of lake benthos except for the oligochaete worms (aquatic earthworms, or Tubificidae), which have not been tested in the laboratory. True midges (chironomids) generally make up the bulk of the benthic biomass in most lakes and ponds (Merritt and Cummins, 1978).

Lindgren's (1960) laboratory tests showed what an important influence access to the bottom sediments has on the survival of benthic fauna exposed to rotenone. Figure 19 shows clearly that when midge larvae had access to the bottom muds, they sustained only a 50% mortality when subjected to a dosage ten times that which killed all midges in a bare aquarium (3.0 ppm as opposed to 0.3 ppm).

Rotenone's immediate effect on benthic animals in lakes and ponds varies, but it does not affect them as drastically as it does plankton. Table M displays the results of thirteen studies on 23 lakes and ponds; in nine of these, the investigators recorded

Table L. Toxicity of rotenone to benthic animals in laboratory bioassays.

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
TURBELLARIA (Flatworms)							
Planaria	0.500	48 hrs.			5% rotenone	100% mortality	Hamilton 1941
Catenula sp.	1.72	96 hrs.	16±1°	20 mg/l total hardness pH 6.6, limed water	Noxfish	LC50	Chandler & Marking 1982
HIRUDINEA (leeches)							
unid. leeches	0.100	48 hours			5% rotenone	90% mortality	Hamilton 1941
	0.100	55 hrs.			5% rotenone	100% mortality	
CONCHOSTRACA							
Estheria mexicanus	0.050	48 hrs.			5% rotenone	90% mortality	Hamilton 1941
	0.050	58 hrs.			5% rotenone	100% mortality	
OSTRACODA							
Cypridopsis sp.	0.340	96 hrs.	16±1°	20 mg/l total hardness pH 6.6 limed water	Noxfish	LC50	Chandler & Marking 1982
Eucypris	0.1	48 hrs.	27-29°	pH 7.2	4.9% rotenone	Minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
MALACOSTRACA							
ISOPODA							
Asellus aquaticus	0.5	6 days	11±1°	pH 7.0 260 mg/l hardness	5% rotenone	30% mortality	Meadows 1973
	1.0	47 hrs.	18±1°		Chem-Fish Special Pro-Noxfish	60% mortality	Lindgren 1960
AMPHIPODA							
Gammarus pulex	2.0	6 days	11±1°	pH 7.9 260 mg/l hardness	5% rotenone	10% mortality	Meadows 1973
Hyallela	0.2	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimal lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952

Table 6 Continued

Organism	Dosage (ppm)	Exposure	Water Temp. °C	Water chemistry	Formulation	Comments	Reference
MALACOSTRACA (cont'd.)							
unid. amphipods	0.500	12 hrs.			5% rotenone	100% mortality	Hamilton 1941
DECAPODA							
Palaemonetes kadiakensis (freshwater prawn)	1.12	96 hrs.	16 ± 1°	20 mg mg/l total hardness pH 6.6 limed water	Noxfish	LC50	Chandler & Harking 1982
Palaemonetes (freshwater prawn)	4.0	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
Cambarus immunis (crayfish)	0.500				5% rotenone	unaffected	Hamilton 1941
Procambarus sp. (crayfish)	10.0	96 hours	20±1°	pH 7.2-7.8 100 mg/l hardness	5% wettable powder	2.5% died	Brown 1973
	75.0	96 hrs.	20±1°	pH 7.3-7.8 100 mg/l hardness	5% wettable powder	5% died	
Orconectes immunis (crayfish)	34.5	24 hrs.	12°	pH 7.2-7.6 40-48 mg/l hardness	Noxfish	LC50	Farringer 1972
	10.8	24 hrs.	12°		Dri-Noxfish	LC50	
	47.2	24 hrs.	12°	pH 7.6-8.0 160-180 mg/l hardness	Noxfish	LC50	
	9.6	24 hrs.	12°		Dri-Noxfish	LC50	
	1.0	96 hrs.	12°	pH 7.2-7.6 40-48 mg/l hardness	Noxfish	LC50	
	0.7	96 hrs.	12°		Dri-Noxfish	LC50	
	1.2	96 hrs.	12°	pH 7.6-8.0 160-180 mg/l hardness	Noxfish	LC50	
	0.4	96 hrs.	12°		Dri-Noxfish	LC50	
EPHEMEROPTERA (mayflies)							
Siphlonurus	1.25	48 hrs.	21-23°	pH 7.0-7.3	5% rotenone	50% mortality	Claffey & Rock 1967
Caenis sp. (mayfly)	0.1	30 hrs.	18±1°		Chem-Fish Special	50% mortality	Lindgren 1960
	0.5				Pro-Noxfish	50% mortality	
ODONATA (dragonflies & damselflies)							
Macromia sp.	1.00	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Harking 1982

Table 6 Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
ODONATA (cont'd.)							
Amphiagrion sp.	2.5	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
Pachydiplax, Iramea	3.5						
Basiaeschna janata	0.22	96 hrs.	22°	pH 7.2 140 mg/l total hardness	"rotenone"	LC50	Watkins & Tarter 1974
Anax	2.25	48 hrs.	21-23°	pH 7.0-7.3	5% rotenone	50% mortality	Claffey & Rock 1967
Agrion	2.6						
HEMIPTERA (bugs) unid. corixids (water boatmen)	1.000	48 hrs.			5% rotenone	40% mortality	Hamilton 1941
unid. notonectids (backswimmers)	0.01	24 hrs.			5% rotenone	unaffected	Hamilton 1941
	0.025	24 hrs.				unaffected	
	0.100	24 hrs.				50% mortality	
Notonecta sp. (backswimmers)	0.1		18±1°		Chem-Fish Special	29% mortality	Lindgren 1960
	0.5				Pro-Noxfish	72% mortality	
Notonecta sp. (backswimmers)	1.58	96 hrs.	16 ± 1°	pH 6.6 limed water 20 mg/l total hardness	Noxfish	LC50	Chandler & Marking 1982
COLEOPTERA (beetles) Gyrinus sp. (whirlygig beetle)	0.700	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
TRICHOPTERA (caddisflies) Hydropsyche sp.	0.605	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
Hesperophylax sp.	10.4	24 hrs.	12°	pH 7.2-7.6 40-48 mg/l hardness	Noxfish	LC50	Farringer 1972
	5.1				Dri-Noxfish	LC50	

Table L Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
TRICHOPTERA (cont'd.)							
Hesperophylax sp.	15.0	24 hrs.	12°	pH 7.60-8.0	Noxfish	LC50	Farringer 1972
	5.1			160-180 mg/l hardness	Dri-Noxfish	LC50	
	3.4	96 hrs.	12°	pH 7.2-7.6	Noxfish	LC50	
	3.2			40-48 mg/l hardness	Dri-Noxfish	LC50	
	2.5	96 hrs.	12°	pH 7.6-8.0	Noxfish	LC50	
	3.6			160-180 mg/l hardness	Dri-Noxfish	LC50	
DIPTERA (true flies)							
Culex, Aedes, Anopheles (mosquitoes)	2.0	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
midges	3.0	18 hrs.		pH 8.3-8.7	derris	10% mortality	Fellton 1940
	6.0	18 hrs.		250-350 mg/l hardness	(5% rotenone)	5% mortality	
	12.0	18 hrs.				25% mortality	
	2.0					55% mortality	
	3.0					50% mortality	
	4.0	46-52 hrs.				90% mortality	
	5.0					95% mortality	
	6.0					100% mortality	
unid. midges	0.31	48 hrs.	27°		Pro-Noxfish	LC50	Brooks 1961
Tendipes crassicaudatus, T. plumosus (midges)	0.25	48 hrs.	27°		cubé	LC50	Wright 1957
	0.10				Noxfish	LC50	
	0.33				Pro-Noxfish	LC50	
Tendipes decorus (midges)	1.0	96 hrs.		pH 5.9-6.1 5-154 mg/l m.o. alkalinity		seriously affected	Taube et al. 1954
Chironomus (midges)	0.1	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952

Table L Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
DIPTERA (cont'd.)							
unid. phantom midges	1.13	48 hrs.	27°		Pro-Noxfish	LC50	Brooks 1961
Chaoborus punctipennis (phantom midges)	0.65	48 hrs.	27°		cubé	LC50	Wright 1957
	1.07				Noxfish	LC50	
	1.13				Pro-Noxfish	LC50	
Chaoborus astictopus (winter larvae)	1.0				derris (5% rotenone)	98% mortality	Negherbon 1959
	0.5					97% mortality	
Palpomyia sp. (biting midges)	3.0	36 hrs.	18±1°		Chem-Fish Special	80% mortality	Lindgren 1960
unid. chironomid	0.5	32 hrs.	18±1°		Pro-Noxfish	50% mortality	
ACARI (water mites)							
unid. water mites	0.0166	4 days			5% rotenone	unaffected	Hamilton 1941
	0.05					43% mortality	
MOLLUSCA (snails & clams)							
Physa halei (snail)	0.1	48 hrs.			5% rotenone	20% mortality	Hamilton 1941
Lymnaea stagnalis (snail)	1.0	3.5 days			5% rotenone	70% mortality	Hamilton 1941
	0.5					30% mortality	
Physa pomilia (snail)	4.00	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
Oxytrema catenaria (snail)	1.75					LC50	
Heliosoma sp. (snail)	7.95					LC50	
Elliptio buckleyi (Buckley's filter clam)	2.95					LC50	
Elliptio complanata (flattened filter clam)	2.00					LC50	

Table L Continued

Organism	Dosage (ppm)	Exposure	Water temp. °C	Water chemistry	Formulation	Comments	Reference
MOLLUSCA (cont'd.)							
Corbicula manilensis (Asiatic clam)	7.50	96 hrs.	16±1°	pH 6.6 20 mg/l total hardness limed water	Noxfish	LC50	Chandler & Marking 1982
Physa (snail)	4.5	48 hrs.	27-29°	pH 7.2	4.9% rotenone	minimum lethal dose, weakest concentration producing a kill exceeding 25%	Zischkale 1952
Helisoma (snail)	3.5						
Viviparus viviparus (snail)	1.0	110 hrs.	18±1°		Chem-Fish Special Pro-Noxfish	unaffected	Lindgren 1960
	5.0	57 hrs.	18±1°			50% mortality	
Planorbis planorbis (snail)	10.0	47 hrs.	18±1°		Chem-Fish Special Pro-Noxfish	unaffected	Lindgren 1960

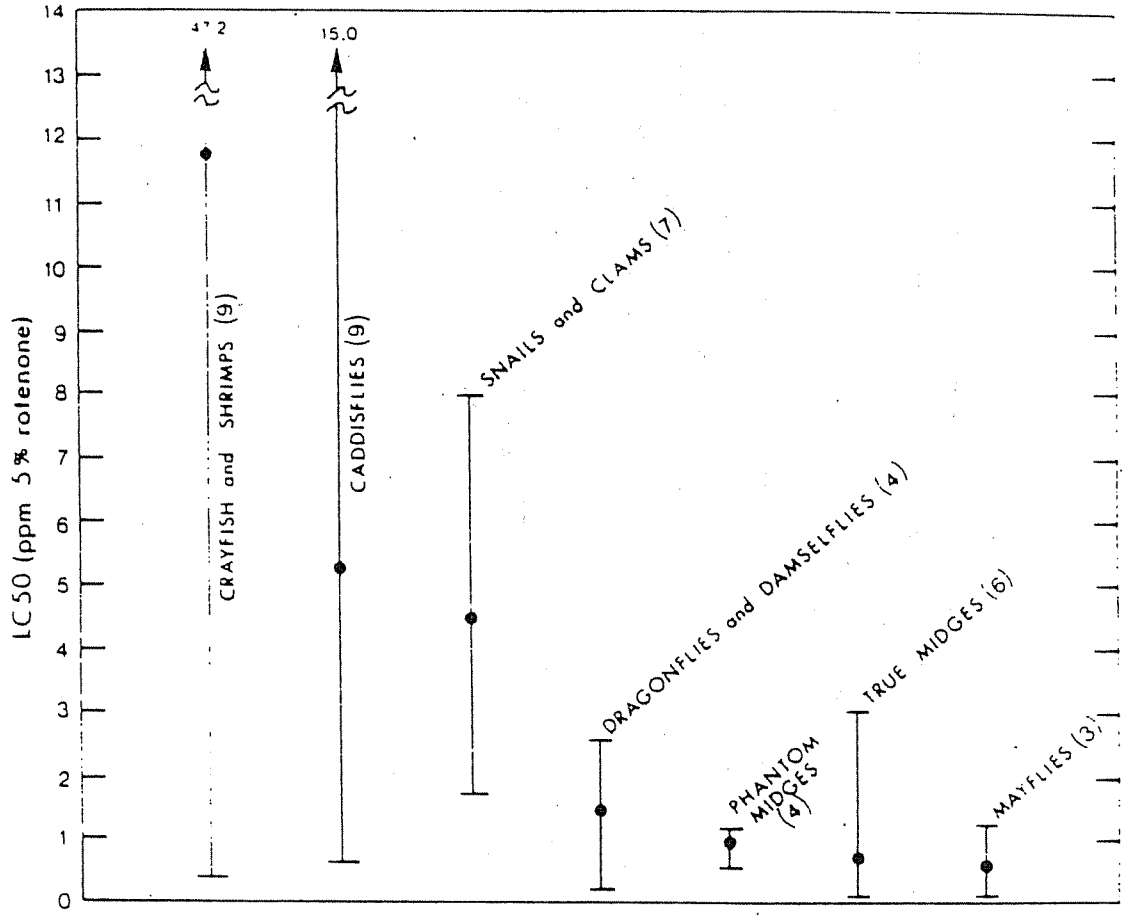

Figure 18 Mean LC50's of rotenone formulation for various groups of lake and pond benthos. Data are drawn from Table 13. Vertical bars represent the range of LC50's found in the literature. Numbers in parentheses represent the number of data points (tests) used in computing the means.

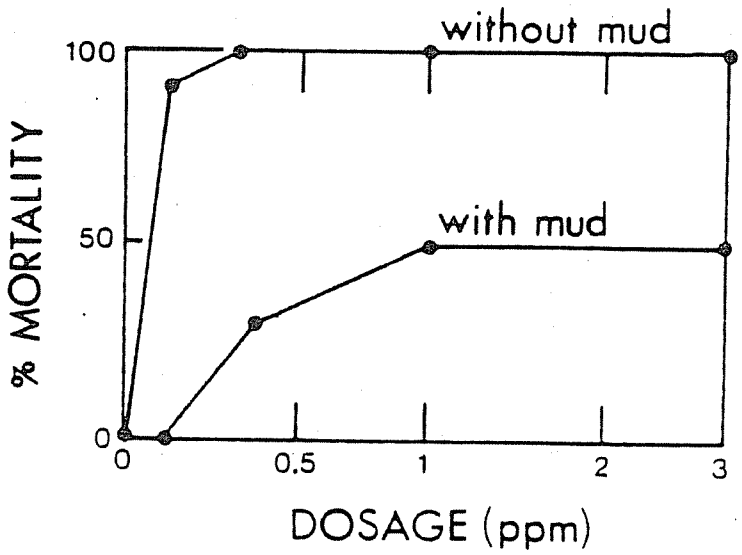

Figure 19 Effect of bottom muds on the survival of midge larvae (Chironomus plumosus) in aquariums subjected to various dosages of rotenone (ChemFish Special). Source: Lindgren 1960.

Table M Short and long-term effects of rotenone treatment on the benthos of various lakes and ponds.

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of benthic abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotenone levels	Comments	Reference
Ponds C & D Missouri	0.1 each	0.5 (Noxfish)	3 mo.	1 year	none	0% within 14 days	immediate	NO	none	no significant increase	-ponds heavily vegetated, muddy -results compared with untreated control ponds	Houf & Campbell 1977
Pond E Missouri	0.1	2.0 (Noxfish)	3 mo.	1 year	none	0% within 14 days	immediate	NO	none	no significant increase		
Pond I Georgia	0.1	2.0 (Pro-Noxfish)	1 day	69 days	none	29.5%[a] within 7 days	<37 days	NO	none	121% (69 days later)	-few aquatic plants -only significant decreases in Caenis (mayfly) and 5 genera of Odonata -results compared with untreated control ponds	Burress 1982
Pond J1 Georgia	0.1	5.0 (Pro-Noxfish)	1 day	69 days	none	59.0%[b] within 7 days	<37 days	NO	none	223% (69 days later)		
7 lakes Finland	2-11	up to 0.8 (synergized 2.5% rotenone)	1 year	4 years	perch	---	---	NO	brown trout, rainbow trout (~9 mo.)	0-400% (1-4 yrs later)	-"after the poisoning, the bottom animal density increased in all lakes."	Tuunainen 1970
Bug Lake Wisconsin	11	2.5 (Pro-Noxfish)	3 mo., 24 days	2 years	shiners, minnows, LM bass, pumpkin-seed, rock bass	---	---	NO	brook trout, (7 mo.) fathead minnow (1 yr., 7 mo.)	---	-no significant change in density of dipterans, oligochaetes, Odonata, and gastropods -trichopterans in shallow water never completely recovered, but this not due to rotenone	Serns 1979

Table M Continued

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of benthic abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotenone levels	Comments	Reference
Bill's Lake New Brunswick, Canada	11	0.25 (derris (5% rotenone))	2 days	9 days	eels, lake chub, perch, SM bass, stickle-back	6% within 8 days	---	---	---	---	-rotenone had "no apparent effect on this community of bottom organisms with the possible exception of Hyallela knickerbockeri."	Smith 1940
Potter's Lake, New Brunswick, Canada	113	0.5 (derris (5% rotenone))	2 months	11 months	eels, sucker, brown bullhead, SM bass, pickerel, perch, sunfish	0% within 10 days	Immediate	YES	brook trout (~11 months)	no significant increase	-Chaeoborus killed in large numbers, as well as an unidentified leech.	Smith 1941
Velká Arazimova Czecho-slovakia	0.1	"Lonchocarpus extract"	2 months	2 years, 3 months	perch, roach, bittering, carp	58% within 3 weeks	<2 months	---	carp, LM bass, roach bream (1 month-2 years)	170% (1 year later)	-"the mass development of the bottom fauna was caused by exceptional supply of food from the water column to the bottom."	Lellák 1965
Emmaline Lake Colorado	3	"rotenone" 1.0 (Pro-Noxfish)	3 months	4 years	brook trout	---	---	---	cutthroat trout (3 years, 9 months later)	~500% (4 years later)	-small chironomids replaced by larger ones in absence of fish	Walters & Vincent 1973 Wrenn 1965

Table M Continued

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of benthic abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotenone levels	Comments	Reference
Carls Lake Minnesota	110	3.0 (Chem-Fish, 2 treatments)	1 year, 3 months	2 years	black bullhead	---	---	NO	LM bass, walleye, channel catfish, brown trout, pike, bluegills, black bullhead (within 1 year)	no significant increase	-"rotenone... might have caused a temporary reduction in some species and in the total number of macroinvertebrates..."	Bandow 1980
Middle Pond Montana	20	0.7 (Chem-Fish Special)	1 year, 10 days	9 months	LM bass, black crappie, perch, sucker, carp, black bullhead	42% within 6 days	<1 month	---	---	274% (37 days later)		Wollitz 1962
West Pond Montana	13	0.7 (Pro-Noxfish)	1 year, 13 days	9 months	perch, bluegill, LM bass	71% within 7 days	40 days	---	---	no significant increase		
Third Sister Lake Michigan	10	0.5 (derris (5% rotenone))	2 years	1 year 1 month	bluegill, LM bass, yellow bullhead, shiners	23%[c/] within 3 weeks	<1 month	---	none	~100% (8 months later)	-almost all Chaoborus larvae killed -leeches, aeschnine dragonflies severely affected	Ball & Hayne 1952

Table M Continued

Test water location	Surface acreage	Dosage (formulation) ppm	Length of study before rotenone	Length of study after rotenone	Important fish present before rotenone	Immediate reduction of benthic abundance	Time to recover to former abundance levels	Any species fail to reappear	Fish introduced after rotenone (release date)	Increase in benthic abundance from pre-rotenone levels	Comments	Reference
Irwin Lake Michigan	10	0.55 (5% emulsified rotenone)	3 days	1 year, 2 wks.	perch	10% within 4 days	---	NO	brook trout (1 year later)	---	only Chaoborus significantly affected	Tebo et al. 1954
Deaming Lake Minnesota	13	0.5 (derris root)	9 days	11 days	---	0% within 11 days	immediate	---	---	---		Hooper 1948

a/ Actual decrease was 66.5%, but a 37% decrease occurred in the untreated control pond during the same time; decrease due to rotenone was found by subtraction (66.5% - 37% = 29.5%).

b/ Actual decrease was 96%. See note a/ above.

c/ Authors did not mention a decrease, but their data show a decrease of 23% when compared to the mean decreases in May-June for the three years when the lake was not poisoned.

benthic abundance within three weeks or less of treatment. These data were either compared with previous bottom grabs in the same lake, or with untreated "control" waters.

There is no clear correlation between rotenone dosage and the number of benthic animals lost shortly after treatment. The factors that most likely influenced the varied results were the differing environmental conditions (especially the amount of submerged vegetation and the bottom type) in the lakes and ponds tested. For example, Houf and Campbell (1977) reported no loss of benthos following a heavy application of 2 ppm Noxfish, but their results may be influenced by the fact that their experimental ponds were heavily vegetated and had very muddy bottoms. Both these factors play important roles in detoxifying rotenone and providing a safe haven for benthic animals. The same dosage (2 ppm) in a similar-sized pond with very little aquatic vegetation destroyed almost 30% of the benthos when compared with the untreated control pond (Burress, 1982). Unfortunately, there are not enough compatible data on these environmental variables from all the studies to fully explain the different results.

Although they did not provide enough quantitative data to be included in Table M, a number of other researchers have reported the short-term effects of rotenone treatment on benthos.

Most have reported that rotenone's impact is mild: Hongve (1977) stated that benthic insects were not affected by a dosage of 0.5 Pro-Noxfish in a Norwegian lake. Neves (1975) found that most benthis invertebrates were not distressed by a 0.6 ppm Noxfish treatment of a lake cove, although some dead mayfly and biting midge larvae appeared in subsequent plankton hauls. After poisoning two Canadian lakes with 0.75 ppm derris, Anderson (1970) concluded that benthic oligochaetes, dipterans, caddisflies, and damselflies appeared unaffected by the rotenone; leeches and snails, however, showed high mortalities. Cushing and Olive (1957) reported that oligochaetes were not affected by 1.0 ppm derris in Smith Lake, Colorado, and that reductions in the midge larvae were apparent for only three days following poisoning. Wright (1957) found that 1 ppm Noxfish and Pro-Noxfish did no harm to midge larvae. Zilliox and Pfeiffer (1960) found that rotenone products at 0.5 ppm did not adversely affect the fish-food organisms in New York lakes.

Some authors have reported drastic reductions in benthos following rotenone: Berzins (1958) found that 0.5 ppm rotenone destroyed most of the benthos of two lakes in southern Sweden. Oglesby (1964) reported that a freshwater polychaete, *Nereis limnicola*, was almost entirely exterminated following a 0.5 ppm treatment of Lake Merced, California, with 5% rotenone.

Taube et al. (1954) documented catastrophic reductions of benthos in five Michigan lakes treated with Fishtox (a 5% emulsifiable

liquid) and one treated with 1.7 ppm emulsifiable rotenone. Even a year after treatment, benthic animal density was down 73% - 97% over previous levels. These lakes remained toxic to fish for an unusually long time - between 19 and 33 months after treatment, even though qualitative tests for rotenone proved negative. The authors therefore suspected that their emulsions had been contaminated with a chemical dispersing agent which was responsible for both the extended toxicity and the benthic kill.

Susceptibility of Different Benthic Animals - Laborartory tests have shown that certain types of benthic fauna are more tolerant of rotenone than others. Research in the field has generally corroborated these laboratory findings. Crayfish proved highly tolerant in Bluewater Lake, New Mexico (Huntingdon, 1956), where they were not affected by 1.5 ppm. Lindgren (1960) noted that the genus *Cambarus* was very tolerant of rotenone, and Boccardy and Cooper (1963) reported that crayfish were unaffected in a Pennsylvania stream treated with rotenone. Dead crayfish were reported on the bottom of Liberty Lake, Washington following rotenone treatment (Funk, WSU, pers. comm.).

Gastropods (snails), also shown by laboratory tests to be relatively tolerant, have survived rotenone treatments in the field as well (Smith, 1941; Hooper, 1948; Serns, 1979), although Anderson (1970) reported that snails were among the first benthic animals to show high mortality following a 0.75 ppm treatment in a Canadian lake, and Smith (1941) noted disappearance of a snail, *Campeloma decisum*, after rotenone.

While no laboratory tests are available for comparison, investigators in the field have usually cited oligochaetes (aquatic earthworms, Tubificidae) as being among the most tolerant benthic organisms (Cushing and Olive, 1957; Anderson, 1970; Hooper, 1948; Serns, 1979; Bandow, 1980; Lindgren, 1960), with only one author reporting large kills of oligochaetes following rotenone poisoning (Wollitz, 1962).

Mayfly larvae, shown in laboratory tests to be very sensitive, have been killed in large numbers in several lakes (Neves, 1975; Burress, 1982) while other benthic animals were unaffected or reduced at a lesser rate. Midge larvae (chironomids) also proved fairly sensitive to rotenone in the laboratory (see Figure 18), and field investigators have reported heavy losses following lake and pond treatments (Bandow, 1980; Wollitz, 1962). Anderson (1970), Serns (1979) and Taube et al. (1954), did note that dipteran larvae (largely midges) were unaffected by rotenone treatments.

Leeches were not extensively tested in the laboratory, but Brown and Ball (1943a), and Anderson (1970), Smith (1941), Ball and Hayne (1952), and Meehean (1942) all reported them to be very sensitive to rotenone.

The larval form of the phantom midge is unusual for insects in that it is largely planktonic (Merritt and Cummins, 1978); without the protection of the bottom sediments, and in view of its relatively high sensitivity in the lab (see Figure 18), it might be concluded that they would suffer heavy losses in poisoned lakes. This has been reported in at least four cases (Ball and Hayne, 1952; Smith, 1941; Meehean, 1942; Taube et al., 1954). The latter authors recorded an 82% reduction in *Chaoborus* within five days of poisoning on a Michigan lake. Contradictory reports have come from Hongve (1977) and Wright (1957), both of whom noted chaoborid larvae surviving rotenone treatments in large numbers.

Effects of Insect Emergence - Only one study (Houf and Campbell, 1977) has addressed the direct, short-term effects of rotenone treatment on the emergence of aquatic insects. These authors found no differences in emergence patterns between treated and untreated ponds, and concluded that rotenone at 0.5 ppm and 2.0 ppm did not interfere with insect emergence.

Long Term Effects - Recovery of the Benthic Community - In the eleven studies that quantitatively followed benthic abundance over the long term (i.e., all research cited in Table M except Smith, 1940 and Hooper, 1948), benthos recovered to at least prerotenone levels of abundance at some time after poisoning. However in one of these studies (Serns, 1979), "recovery" was somewhat ambiguous; Serns reported that caddisfly larvae at a shallow-water sampling site never reached their former levels, but he blamed sampling variance and subsequent fish introductions rather than the rotenone itself.

Table M shows the results of six studies in which bottom grabs were taken often enough to determine how long recovery took. In two cases (Houf and Campbell, 1977; Smith, 1941), there was never a reduction in total benthic abundance following poisoning, so recovery was essentially "immediate". In the remaining four studies (representing six bodies of water), where between 23% and 71% of the benthic fauna was initially destroyed, recovery took between 1 and 2 months. Schnick (1974) concurred with this stating, after a review of the literature to date that: "benthic organisms reach equilibrium in a few months after treatment".

In many cases, the benthic fauna not only repopulated the lakes following rotenone, but their numbers increased dramatically over pretreatment levels. Table M shows that this occurred in 6 of 10 studies (13 of 18 test waters).

In four of the six studies where benthos increased significantly (Tuunainen, 1970; Wollitz, 1962; Ball and Hayne, 1952; Walters and Vincent, 1973), the authors claimed that reduced fish predation was the overriding cause.

A loss of predatory fish cannot explain the huge increases noted by Burress (1982), since his experimental ponds never contained fish. Burress himself does not venture a guess, but Lellak (1965) has a hypothesis which may explain the post-rotenone explosion of benthos when fish are not a factor. While admitting that the increase in bottom animals in Velka Arazimova was due in part to the elimination of predatory fish, Lellak claims that the most important factor was the "rain" of dead plankton that occurred shortly after poisoning. On reaching the lake bottom, this formed a new supply of food for the benthic fauna. Lellak supports this hypothesis by pointing out that in bottom areas of untreated ponds closed off to fish, benthic biomass doubled; but in poisoned ponds, the biomass increased sometimes 50-70 fold, or definitely more than would be expected as a result of merely removing the fish.

While this nutrient "rain" undoubtedly boosts benthic production, Walters and Vincent (1973) noted that in Emmaline Lake, Colorado, this increase was only temporary; the excess of bacteria and plankton that accumulated there after poisoning was soon depleted by the growing population of benthic animals.

Disappearance of Species - Smith (1941) reported that the snail *Campeloma decisum* never reappeared in bottom grabs on Potter's Lake, Canada as long as 11 months after poisoning. In the other five studies in which data were suitably detailed for analysis (Houf and Campbell, 1977; Burress, 1982; Serns, 1979; Bandow, 1980; Tuunainen, 1970), all taxa present before rotenone reappeared in samples after rotenone.

Effect on Species Diversity - Species diversity has traditionally been used as a monitor of benthic community stability. Pollution and other environmental disturbances tend to produce a community that is rich in terms of total benthic abundance, but poor in terms of the number of species.

Houf and Campbell (1977), Burress (1982) and Bandow (1980) are the only investigators who have used a quantitative diversity index (Wilhm and Dorris, 1968) to thoroughly examine the long-term effects of rotenone on the species diversity of benthic communities. Houf and Campbell (1977) reported that neither 0.5 ppm nor 2.0 ppm dosages of rotenone changed benthic diversity (d) in their experimental ponds. Burress (1982) noted pronounced reductions in diversity after poisoning ponds with 2.0 and 5.0 ppm rotenone. Diversity returned to prerotenone levels 69 days later in the pond given the lighter dosage, but in the heavily poisoned pond, benthic diversity was still reduced at that time. There was, however, a "strong trend toward recovery". Bandow's (1980) results are somewhat complicated by a winterkill, but post-rotenone diversity on Carls Lake, Minnesota was the same or greater than before treatment.

Changes in Community Structure - In addition to increases in total benthic standing crop following recovery from rotenone treatment in several lakes, some investigators have reported increases in the numbers of particular benthic animals within the community.

Oligochaete worms increased dramatically after poisoning in the lakes studied by Hooper (1948), Cushing and Olive (1957), Lellak (1965), and Bandow (1980). In these cases, oligochaete worms were not initially affected by the rotenone. Wollitz (1962), saw a doubling of the tubificid worm population in Middle Pond, Montana even after a drastic initial reduction. In all these cases, the shift was temporary; populations had restabilized at their former levels before the studies ended.

Aquatic snails and clams increased in several lakes following poisoning: Wollitz (1962) reported that the snails *Gyraulus* and *Lymnaea* increased tenfold over prerotenone numbers, while *Physa*'s population doubled in a Minnesota lake. Aquatic snails increased in numbers following rotenone in Potter's Lake, Nova Scotia (Smith, 1941). Tuunainen (1970) noted much larger populations of the clam *Pisidium* in most of the seven Finnish lakes he poisoned. It is not clear from the literature whether these shifts to increased numbers of mollusks were temporary or permanent.

The midge population increased dramatically in the two Montana lakes studied by Wollitz (1962) and the Czechoslovokian oxbow poisoned by Lellak (1965). In both these cases, the shifts appeared temporary.

It is tempting to attribute all these shifts in community structure to rotenone tolerance. Oligochaete worms, snails, clams and crayfish are generally regarded as being the benthic animals most resistant to the poison. It may be hypothesized that these groups take advantage of the temporary absence of other more sensitive benthic animals to become dominant. Yet the post-rotenone dominance of midge populations in some instances (Wollitz, 1962; Lellak, 1965) does not fit this hypothesis; not only are midges usually rotenone sensitive in the lab and field tests, but Wollitz recorded a drastic initial reduction of midge larvae before the increase. The elmination of predatory fish may at least partially explain these shifts.

Apart from shifts in numerical abundance of certain benthic animals, only one other change in community structure has been observed following rotenone; Walters and Vincent (1973) found that large-sized midge larvae became dominant after poisoning. This shift has been attributed to a decrease in fish predation.

Fish/Benthos Interactions - When rotenone was used to eliminate fish, benthic animals populations increased in most of the test waters cited in Table M. Most authors credited the sudden reduction in fish predation as the main cause. The "rain" of dead

plankton, bacteria, and epiphyton was also mentioned as a probable catalyst for short-term benthic increases.

When these increases occur, the standing crop of benthos remains at the new, higher level if predatory fish are not restocked. This was demonstrated by Ball and Hayne (1952) when they poisoned Third Sister Lake, Michigan, and purposely avoided restocking so that they could follow the effects. They found that the number of benthic animals doubled; at that point, the benthic community reached a dynamic equilibrium whose limits were determined by factors other than fish predation. Annual cycles of abundance were undisturbed (Figure 20). A doubling of the benthic standing crop following fish removal was also recorded in experimental enclosures on a Swedish lake (Andersson et al., 1978).

Walters and Vincent (1973) ran a similar experiment in Emmaline Lake, Colorado and Figure 20 compares their results with Ball and Hayne (1952). They poisoned the lake's brook trout population and did not stock fish again until almost four years later, near the end of the study. Their results were similar to Ball and Hayne's, with the benthic population increasing about 3.5 times over prerotenone levels. These authors found that, in the absence of fish predation, benthic population regulation at the new, higher level occurred through density-dependent larval mortality.

When fish are restocked into a lake where post-rotenone benthic increases have occurred, the benthic standing crop generally returns to prerotenone levels. Lellak (1965) observed a dramatic increase in pond benthos following poisoning, but two years later (after ther gradual introduction of new fish), both the abundance and biomass of the bottom fauna stabilized within the prerotenone limits (Figure 21).

In what is probably the best and most detailed of the studies, Tuunainen (1970) observed a clear relationship between the bottom animals and fish in seven Finnish lakes; after perch were poisoned with rotenone, benthic diversity increased in all the lakes. This increase was most obvious in the year following poisoning. After releasing new fish, brown trout (*Salmo trutta*) and rainbow trout (*Salmo gairdneri*),into the lakes, the benthic standing crop decreased again, although it remained at a somewhat higher level than before rotenone. Thereafter, benthos increased whenever there was a decrease in fish biomass; in some cases, this increase was even greater than the increase just after poisoning. The typical case of Lake Sahalalmpi is plotted in Figure 21.

While killing all the fish in the test lakes usually resulted in an increase in benthos, there were important exceptions: Table M showed that no increases occurred in the lakes studied by Bandow (1980), Smith (1941), and in one of the ponds studied by Wollitz (1962). Whether or not benthos increases following a fish-kill program depends a great deal on the types of fish killed and their

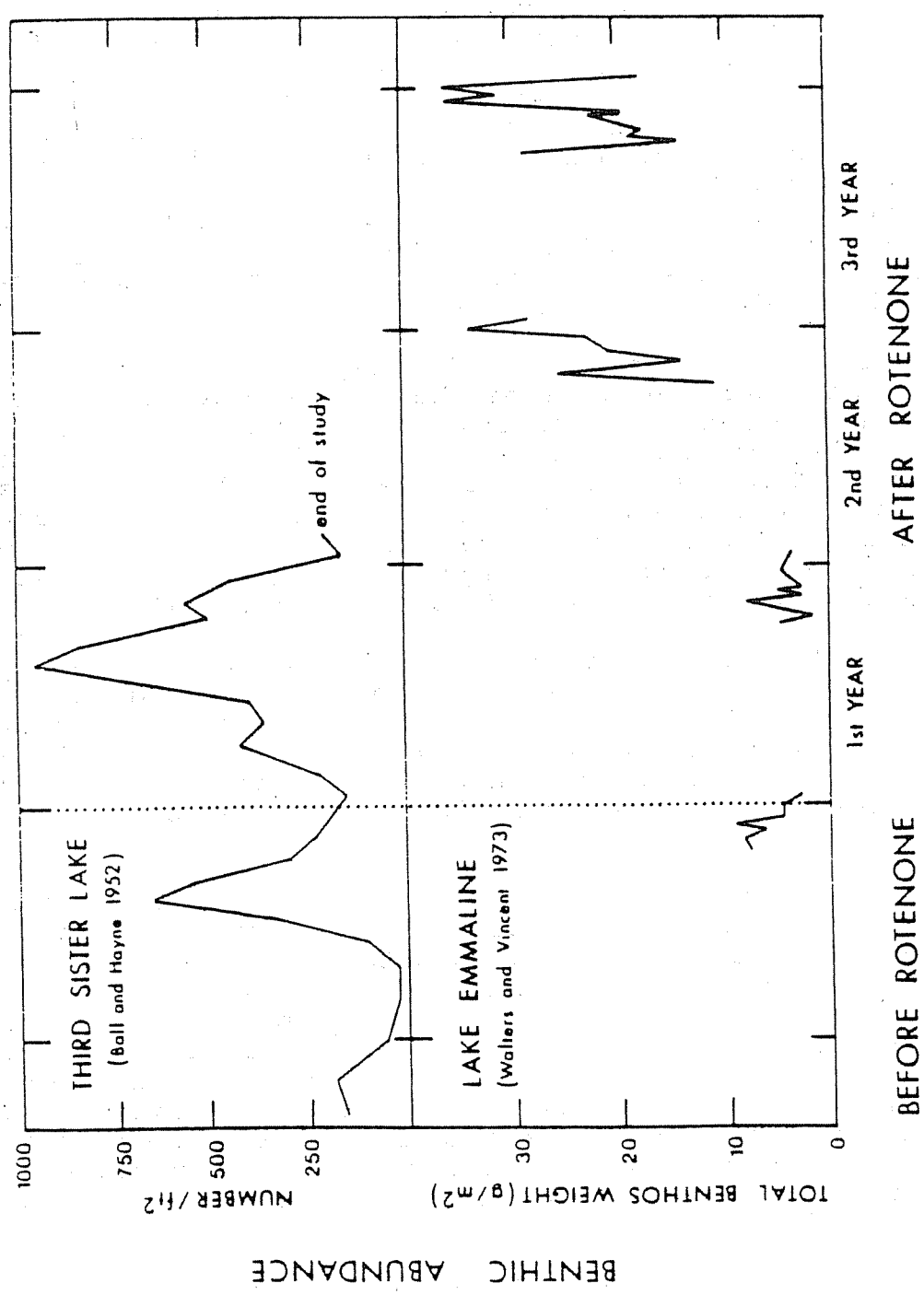

Figure 20 Effect of fish removal on benthos in two lakes where fish were not restocked following rotenone treatment. Dotted vertical line indicates the date of rotenone treatment.

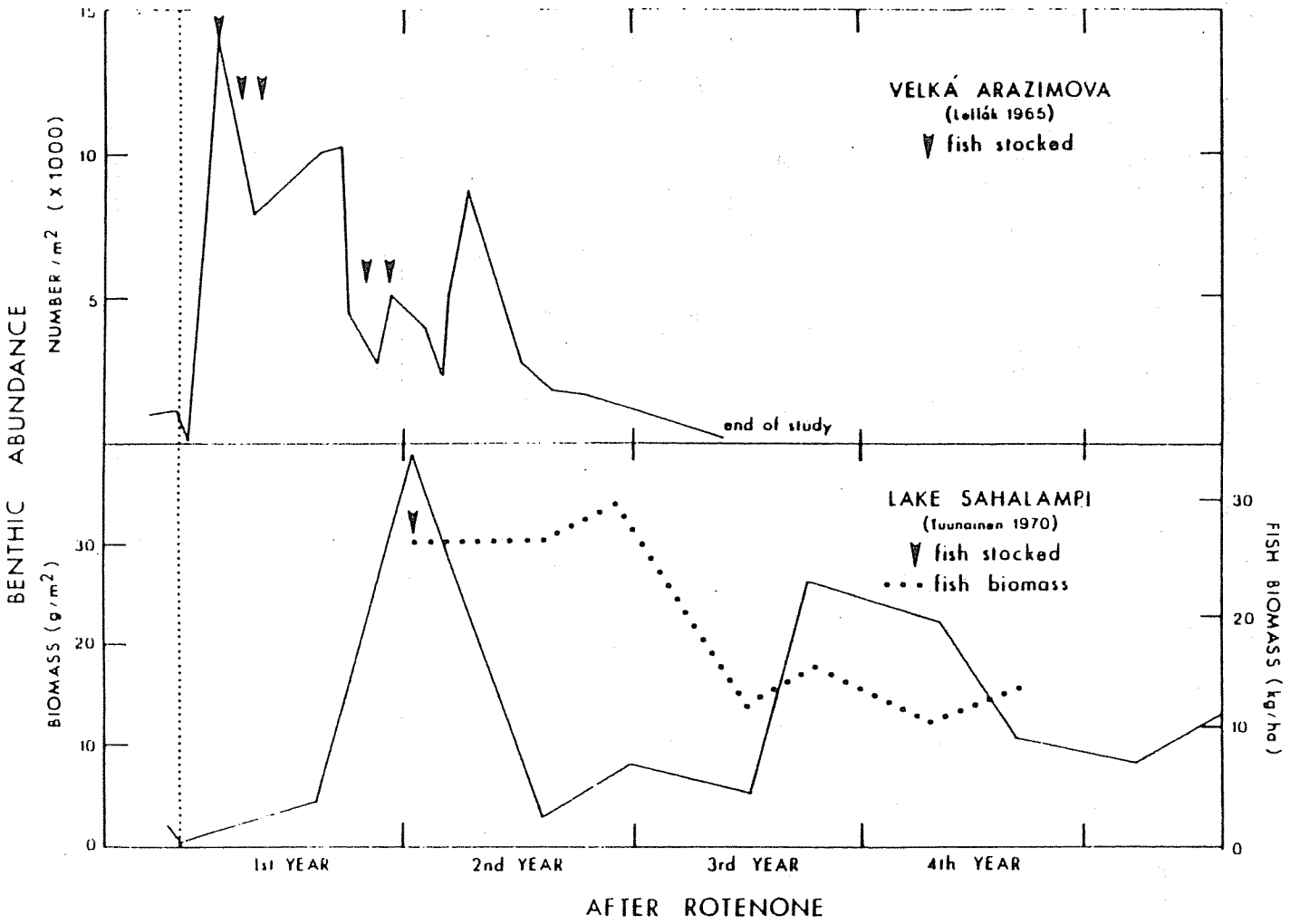

Figure 21 Effect of fish removal and subsequent restocking on benthos in two lakes. Dotted vertical line indicates date of rotenone treatment.

reliance on the lake's bottom animals as a food source. This factor may explain why the standing crop of benthos in Carls Lake, Michigan (Bandow, 1980) was not significantly affected following rotenone; Bandow reported that the most common fish prior to poisoning was the black bullhead (*Ictalurus melas*), and these were heavily dependent on *Daphnia* for food.

No such explanation is readily apparent for the other two cases (Smith, 1941; Wollitz, 1962) in which benthos was unaffected. There are other factors that may influence the way in which a benthic community reacts to fish removal; Tuunainen (1970) claims that lake size alone may be such a factor. With respect to rotenone removal of fish, he states that "small lakes or ponds with quite a small water volume are more susceptible to environmental changes than large ones". Other limnologists have concurred with this statement in regard to fish introductions (Li and Moyle, 1981; Magnuson, 1976). It is probably no coincidence, then, that the two largest lakes studied (Carls Lake and Potter's Lake) showed no long-term changes in the benthic standing crop after fish were killed with rotenone; much smaller lakes always exhibited large increases; with the exception of West Pond (Wollitz, 1962).

A final factor that probably influences the magnitude of fish/benthos interactions in rotenone-poisoned lakes is trophic state. Tuunainen (1970) claims that the effect of removing and restocking fish on the benthic community is much greater in oligotrophic lakes than in eutrophic lakes. As evidence, he compared his oligotrophic Finnish lakes with those eutrophic ponds poisoned by Lellak (1965): the magnitude of the benthic response to fish stocking and changes in fish biomass were dramatic in the nutrient-poor Finnish lakes, while in the eutrophic waters, the bottom fauna restabilized after the initial increase. Li and Moyle (1981) have confirmed that the impact of fish introductions is much greater, and more unpredictable, in oligotrophic lakes than in eutrophic ones.

Emmaline Lake, Colorado was one of the smallest lakes studied, and is also a highly oligotrophic alpine lake: benthos increased most dramatically in Emmaline Lake (~350%) following fish removal, possibly illustrating the combined influence of lake size and trophic state.

Apart from these quantitative changes in the benthic community, only one other aspect of fish/benthos interactions in rotenoned lakes has been studied: Walters and Vincent (1973) noted a shift to larger-sized midge larvae in the absence of fish. Unfortunately, their study did not run long enough following restocking to determine if the situation reversed with fish present.

Effects on Stream Benthos - Although WDW very rarely uses rotenone in running waters, brief mention should be made of the published papers on rotenone's effect on stream benthos. Bridges and Cope (1965), Claffey and Ruck (1967), and Engstrom-Heg et al. (1978) have all performed laboratory bioassays with rotenone on stream insects. Rotenone's short- and long-term effects on stream benthos in the field were investigated by Dexter (1965), Swan (1965), Binns (1967), Cook and Moore (1969) and Helfrich (1978).

In general, these studies demonstrated that rotenone has a far more drastic initial impact on stream benthos than on lake benthos. And while stream invertebrate communities do recover from rotenone, it takes more time than in standing water. The three main reasons for the increased sensitivity of stream benthos are:

1) On the whole, stream-dwelling insects themselves are far more sensitive to rotenone than those that live in lakes (Helfrich, 1978; Engstrom-Heg et al., 1978). Considering rotenone's status as a respiratory poison, this stands to reason; most stream invertebrates have very high dissolved oxygen requirements (Engstrom-Heg et al., 1978), and are less tolerant of a wide variety of pollutants than lake-dwelling insects (Hynes, 1970).

2) Stream applications, to be effective in killing fish usually require much higher rotenone concetrations than do lakes (Binns, 1967; Engstrom-Heg et al., 1978).

3) Streams generally provide less of the organic debris and mud that detoxify rotenone and protect lake-dwelling insects (Lindgren, 1960).

Fish

Short-Term Effects - The median lethal concentrations (LC50) of rotenone formulations for a variety of fish are displayed in Table N. More data are available in the literature, but much of the early work followed no standard procedure; dose-effect experiments have been standardized as 24 to 96-hour LC50's (Marking, 1975), and only these data are reported in Table N.

The upper range of the 96-hour LC50's for all species tested was 0.497 ppm. This is a far lighter dosage than the 1.23 ppm mean dosage used in Washington state lakes. Furthermore, dosages of at least 1 ppm and up to 5 ppm are repeatedly recommended for lake treatments nationwide (Schnick, 1974; Spitler, 1970).

Table N Toxicity of rotenone to fish in laboratory bioassays. All dosages are expressed as median lethal concentrations (LC50).

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
AMIIDAE (bowfins)						
bowfin	0.0575	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0300	96 hr.				
ICTALURIDAE (catfishes)						
channel catfish	0.400	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.161	96 hr.				
	0.033	24 hr.	24°	35 mg/l alkalinity pH 7.1	4.85% rotenone powder	Bridges and Cope 1965
	0.029	48 hr.				
	0.028	96 hr.				
black bullhead	0.665	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.389	96 hr.				
brown bullhead (1.0-1.4 inches)	0.247	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish	Hester 1959a
	0.346				cubé (7.3% rotenone)	
	0.410				Pro-Noxfish	
brown bullhead (6-8 inches)	0.844	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish	Hester 1959a
	0.794				cubé (7.3% rotenone)	
	1.033				Pro-Noxfish	
SALMONIDAE (trout and salmon)						
rainbow trout	0.031	24 hr.	13°	pH 7.1 35 mg/l alkalinity	4.85% rotenone powder	Bridges and Cope 1965
	0.027	96 hr.				
	0.028	48 hr.				
	0.0689	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0460	96 hr.				
	0.057	96 hr.	12°	*	Chem-Fish Regular	Howland 1969
	0.057	48 hr.	12°	*		
brook trout	0.0470	24 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.0443	96 hr.				

Table N Continued.

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
brook trout (fingerling)	0.0470	96 hr.	12°	*	Noxfish	Olson and Marking 1975
lake trout	0.0269 0.0269	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
lake trout (fingerling)	0.0269	96 hr.	12°	*	Noxfish	Olson and Marking 1975
Atlantic salmon	0.0350 0.0215	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
chinook salmon	0.0490 0.0369	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
chinook salmon (fingerling)	0.0490	96 hr.	12°	*	Noxfish	Olson and Marking 1976
coho salmon	0.0716 0.0620	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
CATOSTOMIDAE (suckers)						
longnose sucker	0.0672 0.0570	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
white sucker	0.0719 0.0680	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
CYPRINIDAE (minnows)						
goldfish	0.175 0.218 0.242	72 hrs.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a

Table N Continued

Species	Dosage (ppm)	Exposure	Temp. C°	Water chemistry	Formulation	Reference
goldfish (cont'd.)	0.497	96 hr.	12°	*	Noxfish	Marking and Bills 1976
carp	0.081 0.115 0.163	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a
	0.0840 0.0500	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
fathead minnow	0.066	96 hr.	25°	---	2.5% rotenone, 5% cubé extractives, 2.5% sulfoxide	Cohen et al. 1960
	0.400 0.142	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
	0.159 0.200 0.191	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a
golden shiner	0.470 0.620 0.555	72 hr.	21°	pH 7.8-9.5 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a
white amur	0.0630	96 hr.	17°	---	Noxfish	Marking 1972
ESOCIDAE (pikes) northern pike	0.0449 0.0330	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
CENTRARCHIDAE (sunfishes) green sunfish	0.165 0.246 0.238	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a
	0.218 0.141	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976

Table N Continued

Species	Dosage (ppm)	Exposure	Temp. c°	Water chemistry	Formulation	Reference
bluegill	0.026 0.023 0.023	24 hr. 48 hr. 96 hr.	24°	pH 7.1 35 mg/l alkalinity	4.85% rotenone	Bridges and Cope 1965
	0.114	96 hr.	12°	*	Chem-Fish Regular	Howland 1969
	0.179 0.268 0.255	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a
	0.149 0.141	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
largemouth bass	0.147 0.164 0.081	72 hr.	21°	pH 7.8-9.4 30-50 ppm $CaCO_3$	Noxfish cubé (7.3% rotenone) Pro-Noxfish	Hester 1959a
	0.200 0.142	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
smallmouth bass	0.0932 0.0790	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
PERCIDAE (perches)						
yellow perch	0.0920 0.0700	24 hr. 96 hr.	12°	*	Noxfish	Marking and Bills 1976
walleye	0.0165	24 hr.	12°	*	Noxfish	Marking and Bills 1976

* fish were tested under a range of water chemistries; see Lennon and Walker (1964) for laboratory procedures.

Markings and Bill cited four reasons for the apparent discrepancy between recommended field dosages and dosages known to be lethal in the lab:

1) Laboratory results (LC50's) indicate dosages that kill 50% of the fish, whereas the ideal field dosage is one that kills 100% of the target fish.

2) Organisms, particulate matter, and sunlight in natural waters tend to detoxify rotenone faster than in laborartory aquaria.

3) Uniform concentrations are far more difficult to achieve in the field, so that higher dosages are needed.

4) Individual fish of a species may be exceptionally resistant, so that a higher dosage is needed.

Markings and Bill (1976) concluded, along with Burress (1975), that field concentrations should be based on the results of on-site toxicity test rather than on laboratory or field data. Laboratory data can serve as guidelines in selecting field dosages (Gilderhus, 1972).

Susceptibility of Different Fish Species - Laboratory tests can also serve as indicators of the relative susceptibility of different fish species. Figures 23 and 24 display the results of the most thorough study on this subject (Markings and Bill, 1976). Of the twenty species tested under standardized conditions, goldfish (*Cyprinus carpio*) and black bullheads (*Ictalurus melas*) were the most resistant - 10 times as resistant as most other species.

These results are in general agreement with earlier, less detailed studies: Leonard (1939) stated that the least resistant species included the common shiner (*Notropis cornutus*), golden shiner (*Notemigonus crysoleucas*), bluegill (*Lepomis macrochirus*), pumpkinseed (*Lepomis gibbosus*), and brook stickleback (*Culaea inconstans*), while the mudminnow (*Umbra spp.*), and goldfish, (*Carassius auratus*), were the most resistant; Burdick et al. (1955) placed the following fish in order of their increasing resistance: brown trout (*Salmo trutta*), rock bass, (*Ambloplites rupestris*), creek chub (*Semotilus atromaculatus*), smallmouth bass (*Micropterus dolomieui*), common sucker (*Catostomus commersoni*), and brown bullhead (*Ictalurus nebulosus*). Jenkins (1956) ranked the following from least to most resistant: gizzard shad (*Dorsoma cepedianum*), carp (*Cyprinus carpio*), largemouth bass (*Micropterus salmoides*), redear sunfish (*Lepomis microlophus*), black crappie, (*Pomoxis nigromaculatus*), bluegill, white crappie (*Pomoxis annularis*), green sunfish (*Lepomis cyanellus*), warmouth (*Lepomis gulosus*) and black bullhead (*Ictalurus melas*).

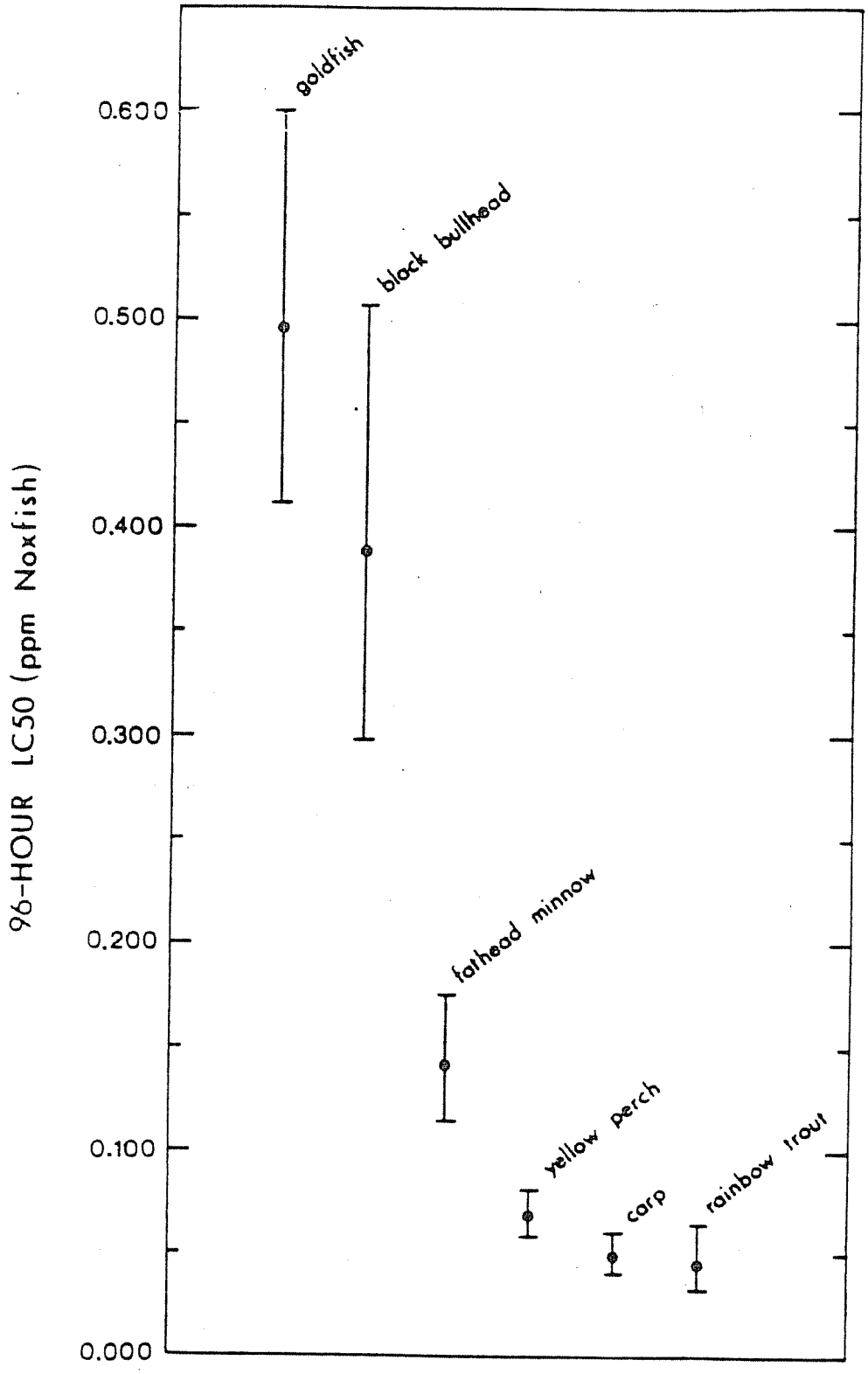

Figure 23 96-hour median lethal concentration (LC50) of Noxfish for several fish held under standardized laboratory conditions. I. Vertical bars represent 95% confidence intervals. Data from Marking and Bills 1976. See Figure 2 for additional data.

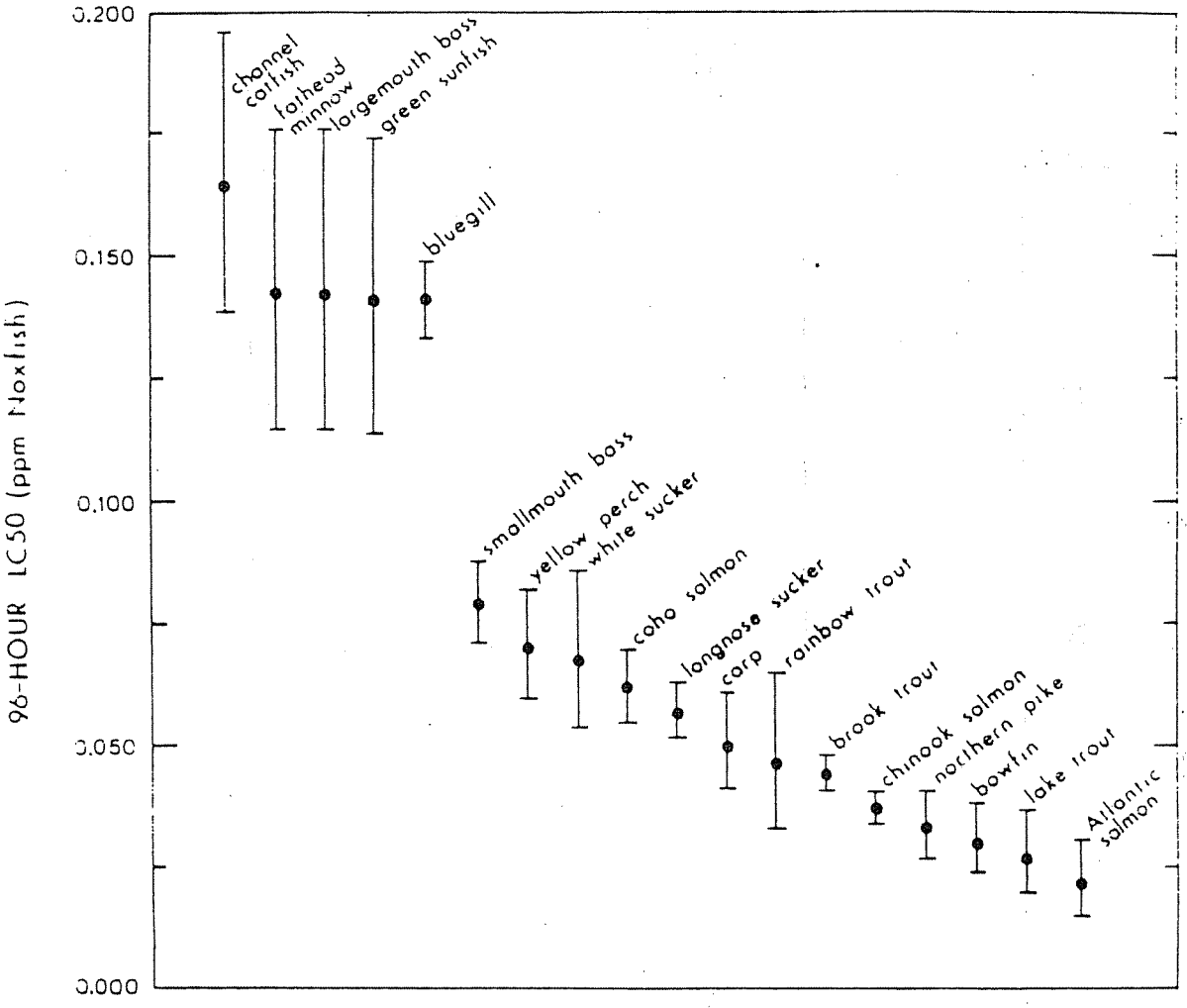

Figure 24 96-hour median lethal concentration (LC50) of Noxfish for several fish held under standardized laboratory conditions. II. Vertical bars represent 95% confidence intervals. Data from Marking and Bills 1976. See Figure 1 for goldfish and black bullhead LC50's.

Effect on Fish Eggs - Table O displays the results of laboratory toxicity tests on fish eggs. All researchers working with salmon eggs found that they were more resistant to rotenone than fry or fingerlings of the same species. Olson and Marking (1975) compared fingerling brook trout, lake trout (*Slavelinus namaycush*), and chinook salmon (*Oncorhynchus tshawytscha*) with eggs of those species; they concluded that the eggs were more resistant. Markings and Bill (1976) found that newly fertilized eggs of rainbow trout were 47 to 106 times more resistant than rainbow fingerlings to Noxfish. The actual degree depended on water hardness. Garrison (1968) reported that salmon eggs were 10 times as resistant to Pro-Noxfish than were salmon fry. He suggested that salmonid embryos would survive a fish-killing dose of rotenone. Leonard (1939) found that eyed brown trout eggs survived a 0.5 ppm dosage of derris powder, but that the fry died as soon as they broke the shell.

Leonard (1939) and Clemens and Martin (1953) reported that problem species have repopulated in lakes where they have been completely poisoned out, and where no illegal stocking or invasion from nearby waters occurred. They suggested that resistant eggs which hatched after detoxification could have been the reason. Some support for this hypothesis comes from Markings et al. (1983), who found that eyed carp eggs were about 50 times as resistant to rotenone as were carp larvae based on LC50 values. Rainbow smelt (*Osmerus mordax*) eggs were about 10 times as resistant as the larval form. Hester (1959b), reported that the LC50's of both carp eggs and fathead minnow eggs were very similar to those obtained with fingerlings of the same species. Either his results were in error, or carp and fathead minnow eggs behave differently than salmonid eggs when exposed to rotenone.

Effect on Non-Target Native Fish - Fish native to Washington state waters are seldom the target of rotenone treatments. It is reasonable to assume that native non-game fish (such as sculpins , suckers, dace, chubs, squawfish and shiners), as well as residual stocked trout, are killed along with target species in a rotenoned lake. Of the nonsalmonid fish native to Washington, only suckers have been tested for their tolerance to rotenone; Figure 24 shows that they succumb to smaller dosages of rotenone than most target species (e.g., perch, sunfish, catfish).

Zilliox and Pfeiffer (1960) reported that native fish in Adirondack Lakes - white suckers, brown bullheads, whitefish (*Coregonus spp.*), and several minnows - were temporarily eliminated along with the non-native target species, usually yellow perch (*Perca flacescens*).

Effectiveness of Treatment - In the past the most common way to judge the effectiveness of a rotenone treatment was on the basis of a "complete kill" of all target fish. Several more or less practical definitions of a "complete kill" have been offered (Clemens and Martin, 1953; Lennon et al., 1970; Zilliox and

Table O Toxicity of rotenone formulations to fish eggs. All dosages are expressed as median lethal concentrations (LC50).

Species	Dosage (ppm)	Exposure	Temp C°	Water Chemistry	Formulation	Reference
rainbow trout (newly fertilized eggs)	5.60 4.42 3.20 2.50	96 hr.	12°	very soft* soft* hard* very hard *	Noxfish	Marking and Bills 1976
chinook salmon (green eggs)	>3.00	96 hr.	12°	**	Noxfish	Olson and Marking 1975
brook trout (green eggs)	3.40	96 hr.	12°	**	Noxfish	Olson and Marking 1975
lake trout (green eggs)	>1.00	96 hr.	12°	**	Noxfish	Olson and Marking 1975
carp (newly fertilized eggs)	0.091 0.178	192-216 hr.	24°	---	Noxfish Pro-Noxfish	Hester 1959b
carp (eyed eggs)	0.025	96 hr.	12°	soft*	Noxfish	Marking et al. 1983
rainbow smelt (eyed eggs)	0.015	96 hr.	12°	soft*	Noxfish	Marking et al. 1983
fathead minnow (newly fertilized eggs)	0.142 0.233	216-247 hr.	21°-24°	---	Noxfish Pro-Noxfish	Hester 1959b

* hardness expressed as mg/l $CaCO_3$: very soft, 10-12; soft, 40-44; hard, 160-180; very hard, 290-310.

** eggs were tested under a range of water chemistries; see Lennon and Walker (1964) for laboratory procedures.

Table 0 Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to birds.

Animal	LD50	Formulation	Reference
White rock chickens	6 ml/kg	Chem-Fish Regular Noxfish	Brooks 1961
	8 ml/kg	Chem-Fish Special Pro-Noxfish	
Chickens (4-week)	>270 mg/kg	pure rotenone	Cutkomp 1943b
Chickens (5-day)	996 mg/kg	pure rotenone	Cutkomp 1943a
Chickens (5-day)	247 mg/kg	derris extract (25% rotenone)	
Eastern chipping sparrow (nestling)	113 mg/kg	pure rotenone	
Eastern song sparrows (nestlings)	130 mg/kg	pure rotenone	
Eastern robins (nestlings)	195 mg/kg	pure rotenone	
English sparrows (nestlings)	199 mg/kg	pure rotenone	
English sparrows (adults	853 mg/kg	pure rotenone	
pheasants (5-day)	850 mg/kg	pure rotenone	
pheasants (4-week)	1190 mg/kg	pure rotenone	
pheasants (3-4 month)	>1414 mg/kg	pure rotenone	Tucker & Crabtree 1970
prairie horned larks (adult)	450-500 mg/kg	pure rotenone	Cutkomp 1943a
mallards (3-4 month)	>2000 mg/kg	pure rotenone	Tucker & Crabtree 1970

Table O Toxicity of rotenone to amphibians in laboratory bioassays.

Animal	Concentration (ppm)	Exposure	Formulation	Water Chemistry	Comments	Reference
Southern leopard frog larvae (Rana sphenocephala)	0.5	96 hr.	Noxfish	16° C; see Lennon & Walker (1964) for test conditions	LC50	Chandler & Marking 1982
Leopard frog (Rana pipiens)	7.3	24 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness	LC50	Farringer 1972
	7.9	24 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
	4.6	96 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	3.2	96 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
Leopard frog tadpoles (Rana pipiens)	0.1	8-24 hr.	5% rotenone	---	100% mortality	Hamilton 1941
tiger salamander, with gills (Ambystoma tigrinum)	0.017	8-24 hr.	5% rotenone	---	toxic but not necessarily fatal	
tiger salamander, metamorphosed (Ambystoma tigrinum)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
frogs	4.0 mg/kg body weight	---	pure rotenone	---	oral LD50	Haag 1931

Table O Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to animals.

Animal	LD50	Formulation	Reference
Rabbits	1.7 ml/kg	Chem-Fish Special Pro-Noxfish	Brooks 1961
White mice	350 mg/kg	pure rotenone	Kenaga and Allison 1971
Rats	1.5±0.1 ml/kg	Pro-Noxfish	Brooks 1961
	170 mg/kg	cubé (4.7% rotenone) in aqueous solution	Haag & Taliaferro 1940
	132 mg/kg	pure crystalline rotenone	Lehman 1951
	1500 mg/kg	derris	Lehman 1951
	1.5 cc/kg	Chem-Fish Special	Blue Spruce Co. 1973
Guinea pigs	60 mg/kg	pure rotenone	Cohen et al. 1960
	55-60 mg/kg	pure rotenone	Cutkomp 1943b

Pfeiffer, 1956). The last authors gave the following commonly-cited criteria for a complete kill: "Failure of observation, angling and netting for two successive years following reclamation to indicate any species of fish present in a reclaimed pond, except stocked trout, would appear to be a reasonable indication of a complete kill". The authors later excluded native species from this definition, since they frequently reappear within two years even in "successfully" treated lakes (Zilliox and Pfeiffer, 1960).

Judged by the criteria of Zilliox and Pfeiffer (1956, 1960), WDW Biologist Bob Pfeifer stated that is was unlikely that complete kills were acheived in recent years in a number of Seattle-area lakes (Pfeifer, 1985).

Clemens and Martin (1953) pointed out that the only way to be entirely sure of a complete kill is to drain the lake or pond. This has been done on occasion: Cumming et al. (1975) drained a 0.1 acre Arkansas pond following a 2 ppm Noxfish application and found that a complete kill of channel catfish and grass carp had indeed occurred. But Clemens and Martin (1953) drained two ponds after rotenone treatment and found fish in both; one pond had been judged a "complete kill" before draining. On six other ponds which Clemens and Martin had initially termed "complete kills", intensive seining revealed some target fish still present in at least five of them.

In larger lakes, the possibility of ever exterminating 100% of the target fish with rotenone is small, and is probably an unrealistic goal (Klingbeil, 1975). Klingbeil notes that massive efforts to kill the last 0.1% of a target population in Wisconsin are usually followed immediately by illegal stocking of the same or different problem species.

Klingbeil (1975) and Zilliox and Pfeiffer (1960) have disregarded the concept of a "complete kill" altogether, offering another criterion by which to judge the effectiveness of a lake poisoning: the return of quality fishing. This would seem to be far more viable measure for two reasons:

1) the ultimate purpose of most treatments is to produce better fishing, not necessarily to eliminate X number of target fish (Prevost, 1960); and

2) quantifying "better fishing" (in terms of catch-per-unit-effort, CPUE, fingerling growth and survival, etc.) is far more practical than determing a "complete kill". These data are already collected on a yearly basis on virtually all Washington state "trout-only" lakes. Cost-benefit analyses can also be readily applied to these lakes.

Biologists have long been interested in what proportion of the fish poisoned in a lake eventually come to the surface, mostly out of a desire to make population estimates more reliable,(Brown and Ball, 1943b; Carlander and Lewis, 1948; Krumholz, 1950b; Lambou and Stern, 1957). It has been suggested that the decay of unrecovered fish that did not surface, might produce nuisance algae blooms in some lakes (Funk and Moore, 1984). There are five main factors that influence the surfacing of dead fish in rotenoned lakes:

1) Water temperature . Parker (1970) made both laboratory and field tests and found that in warm water, dead fish surfaced much more quickly than in cold water. Bartoo (1977) and Krumholz (1950b) also cited water temperature as a major factor in surfacing rates of rotenone poisoned fish.

2) Water depth . Parker (1970) found that deep water slowed the surfacing of dead warmwater fish.

3) Fish species . Parker (1970) reported that dead bullheads surface more slowly than centrachids (sunfish) and dead minnows faster than either. The data of Kempinger and Christenson (1978) indicate that a greater portion of dead walleye come to the surface compared to other warmwater species.

4) Fish size . Smaller (younger) fish surface at a much slower rate than larger fish of the same species (Parker, 1970; Brown and Ball, 1943b; Kempinger and Christenson, 1978).

5) Presence of aquatic rooted plants . When fish have access to extensive beds of underwater vegetation, they often become tangled and fail to surface after they die (Parker, 1970, Ball, 1945; Zook, 1978).

Parker (1970) found that the following factors, within the limits indicated, did not affect surfacing rate: dissolved oxygen (3.8-13.8 ppm); total alkalinity (40.0-140.0 ppm as $CaCO_3$); pH (7.7-8.5), total hardness (110.00222.3 ppm), transparency (clear - 8 inches), and rotenone dosage (0.5-6.3 ppm 5% dust).

Table P displays the data from rotenoned lakes and ponds in which mark-recapture experiments were made using various fish. In every case except Ford Lake (Ball, 1945), the authors were certain of a complete kill. Also, all authors felt that tagging mortality was insignificant and did not bias the results.

Considering the importance of water temperature in the surfacing of dead fish, it is unfortunate that no temperature data exist for some of the test waters. Figure 25 shows the percentage of fish surfacing within 24 hours of rotenone treatment on lakes and ponds where temperature data were available. Although data from studies involving different warmwater fish species, fish sizes, and lake

Table P Percentage of dead fish surfacing following rotenone treatment in mark-recapture experiments.

Test water, location	Species	Water Temp. (°F)	Depth Range (Ft.)	Time to Surface	% of Dead Fish That Surfaced	Reference
North Lake, Western Washington	Yellow perch	50°	1-35	24 hours 96 hours	15.5% 17.5%	Bartoo 1977
Laboratory & 4 ponds, Ohio	Bluegills (& other centrarchids)	80° 72° 63° 59° 50° 40°	1-15	24 hours 48 hours 72 hours 120 hours 32 days 30 days	95-100% " " " " " " " " " "	Parker 1970
-----	Largemouth Bass	68°	---	60 hours 48 hours 24 hours	96% 91% 62%	Krumholz 1950a
Shoofly Lake, eastern Washington	Largemouth bass	65°	1-12	24 hours	46.6%	Zook 1978
Farm pond, Iowa	Bluegill White crappie Largemouth bass Black bullhead Golden shiner	---	----	120 hours	38% 14% 33% 80% 91%	Carlander and Lewis 1948
Nebish Lake, Wisconsin	Walleye Smallmouth bass Northern pike Yellow perch Rock bass Bluegill Pumpkinseed Green sunfish Largemouth bass Mean (all species)	56°	1-45	24 hours	44.0% 16.6% 17.0% 4.2% 22.4% 24.1% 23.0% 7.0% 25.0% 20.4%	Kempinger and Christenson 1978
Ford Lake, Michigan	Bluegill Brook trout	---	1-33	144 hours	59% 45%	Ball 1945

Test water, location	Species	Water Temp. (°F)	Depth Range (Ft.)	Time to Surface	% of Dead Fish That Surfaced	Reference
Farm pond, Indiana	Green sunfish	---	1-11	24 hours 48 hours 72 hours 120 hours 168 hours 192 hours	70.1% 85.4% 88.0% 90.4% 90.8% 91%	Krumholz 1950b
Farm pond, Indiana	Largemouth bass	---	1-11	48 hours	87.1%	Krumholz 1950b
Barkley Lake, Kentucky	Mostly sunfish, bluegill, largemouth bass	---	---	72 hours	89%	Axon et al. 1979

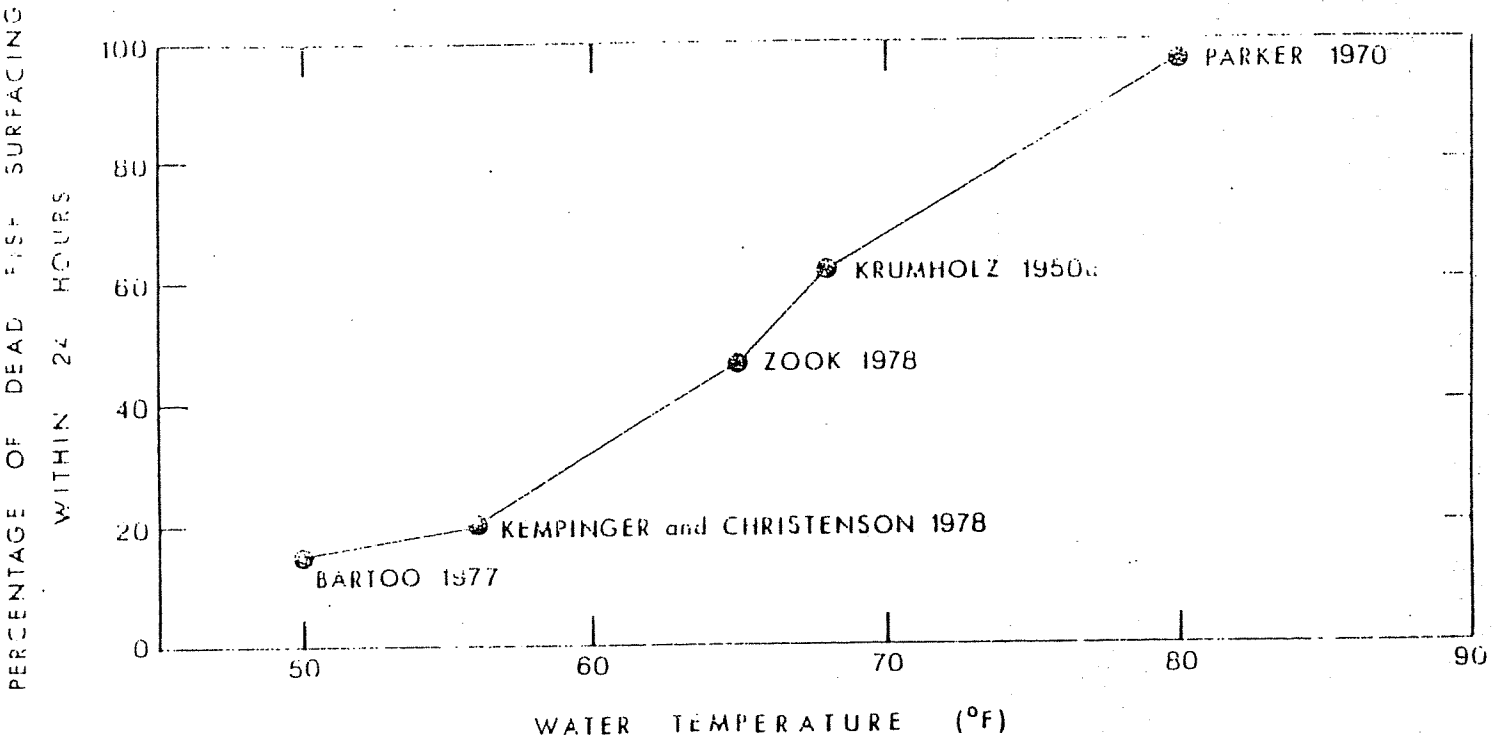

Figure 25. Relationship between water temperature and the percentage of dead fish that surface following rotenone treatment. Data from Table 12.

charateristics have been grouped together, it is still clear that in cold water, a much smaller percentage of dead fish surface than in warm water. Bartoo (1977) found that after the initial 24 hours, few fish surfaced in the relatively cold waters of North Lake, Washington; in the following three days, they were able to recover an additional 2%.

The relationship between fish size and the percentage of dead fish that float to the surface is well demonstrated by the data from Nebish Lake graphed in Figure 26. Regardless of species, the smaller fish showed a tendency to remain on the bottom.

Some investigators have stated that almost all dead fish can eventually be recovered from a lake, since even fish on the bottom will bloat over time and rise to the surface. Hoffman and Payette (1956) report this occurring eight days after rotenone treatment of a San Diego reservoir. This second harvest of bloated fish was actually much greater than the initial collection of dead fish made within five days of poisoning. Brown and Ball (1943a) had SCUBA divers observe individual dead fish lying on the bottom of Third Sister Lake, Michigan; a week after poiosoning, these fish were still on the bottom and decaying.

On Washington lakes, the surface water temperatures at the time of treatment in the fall range from 44°-80° F, averaging 57°-58° F. Based on this mean and Figure 25, we would expect that only about 30% of the dead fish could be recovered. The bulk of the dead fish would never surface, eventually decaying in the lake.

Long Term Effects - Effect on Non-Target Native Fish - No quantitative studies have been made of the long term effects of rotenone poisoning on native fish. Zilliox and Pfeiffer (1960) reported on 12 Adirondack lakes which were rotenoned to eliminate yellow perch, an introduced species: in 1954, all these lakes were judged "complete kills", yet within five years, at least half were repopulated with brown bullheads, white suckers, and several minnow species, all native fish. The author's data indicated that the native species had survived poisoning, rather than merely being reintroduced.

It is reasonable to assume that native, nontarget populations eventually recover in the same way that target fish do: some fish survive either due to individual tolerance (Meyer, 1966; Tompkins, 1953) or, more likely, because a truly "complete kill" has not occurred. Quantitative data on recovery are lacking in the literature.

Complicating the situation is the fact that rotenone target species such as goldfish have a disastrous impact not only on trout, but on other native fish populations as well (Wydoski and Whitney, 1979; Gothschalk, 1966). The question of whether or not rotenone

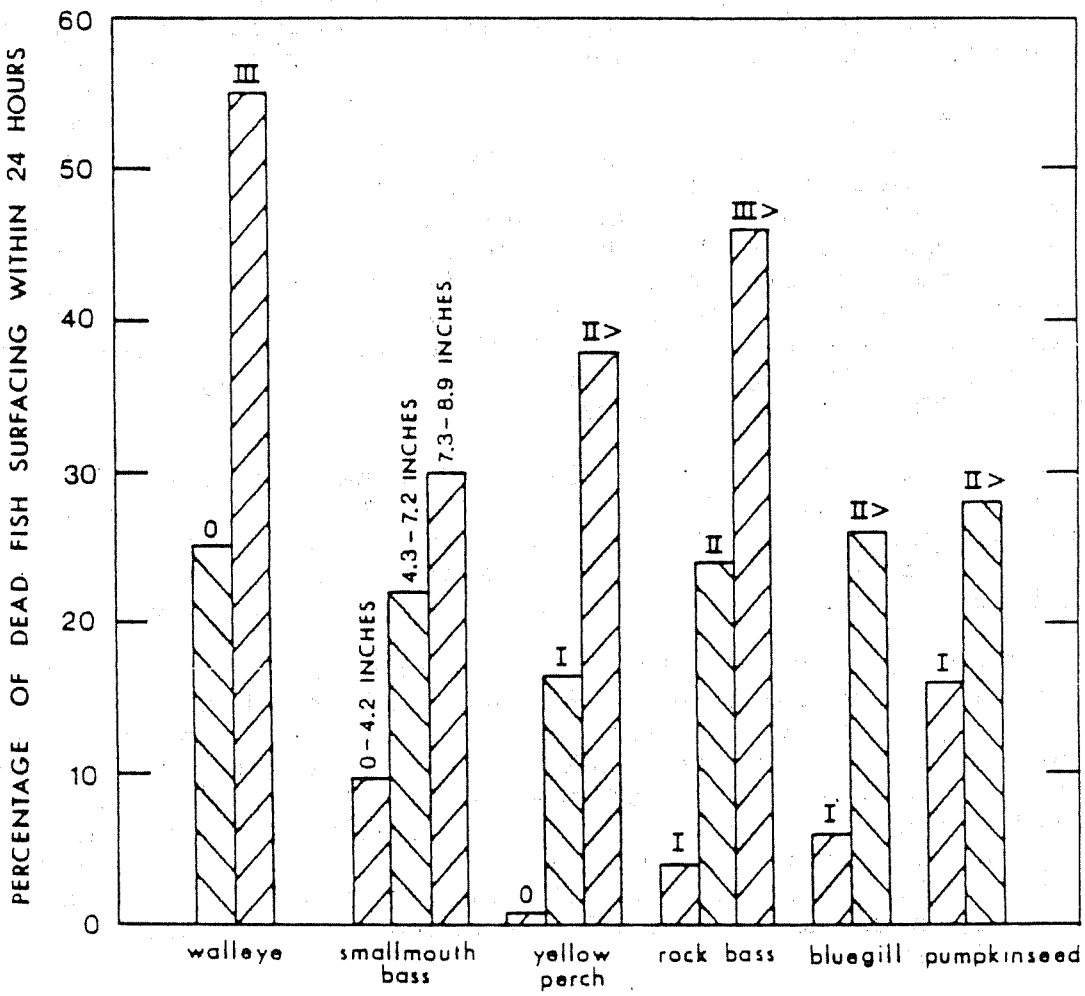

Figure 26 Relationship between fish size (age) and surfacing rate for various species in Nebish Lake, Wisconsin. Roman numerals indicate age group of fish. Surface temperature was 56°F at the time of poisoning. Source: Kempinger and Christenson 1976.

benefits native nongame fish in the long run (by eliminating competitive exotic species) is open to speculation.

Effect on Growth and Survival of Planted Trout - Trout are only restocked in a rotenoned lake after bioassays indicate that the water is completely detoxified, so that rotenone has no direct toxic effect on growth or survival. The indirect, long-term effects of rotenone treatment are increased growth and survival of fingerling trout; this occurs because predators and/or competitors are eliminated, and is the goal of most treatments in Washington state and nationwide (Lennon et al., 1970).

Some authors have cautioned fishery managers not to restock trout while zooplankton and/or benthic populations are still reduced following rotenone (Bennett, 1985; Kiser et al., 1963). Kiser and his colleagues stated that Fern Lake, Washington was stocked by WDW immediately after the lake detoxified about five weeks before zooplankton populations had recovered to prerotenone levels. They noted that successful fingerling stocking depends on an abundance of natural food, and that fish may have been stocked too soon following the spring treatment. There was no followup research on survival or growth.

Almost 80% of Washington state treatments occur in the fall, and trout are restocked the following spring. This far exceeds the time generally required for zooplankton and benthic animals to recover to prerotenone levels. In the case of spring treatments, there are two options: either a prestocking zooplankton sample, or a post-stocking measurement of fingerling growth. The later empirical approach seems more practical and more reliable.

Rotenone Tolerance in Fish - Repeated us of pesticides on crops has led to the well-documented phenomenon of resistant insects that become harder to control. Vertebrates such as fish usually breed too slowly for such resistant populations to develop (Fabacher and Chambers, 1972), but they do occur: Vinson et al. (1963) and Culley and Ferguson (1969) found mosquitofish (*Gambusia affinis*) that had apparently acquired a tolerance to DDT and a wide variety of other pesticides in a heavily-sprayed agricultural area in Mississippi. Hubbs (1963) was the first to theorize that undesirable fish might become tolerant to rotenone, requiring ever more frequent poisonings. Two instances have been reported where fish apparently acquired a tolerance to rotenone through exposure to rotenone or other pesticides: Fabacher and Chambers (1972) found that the insecticide-resistant mosquitofish from Mississippi showed a 1.8-fold tolerance to rotenone over mosquitofish from pesticide free waters. While rotenone was not one of the insecticides used in the area, Fabacher and Chambers demonstrated that heavy, repeated spraying of other organo-chlorine insecticides in the area produced a "cross-tolerance" in these fish.

Orciari (1979) demonstrated an acquired tolerance due to repeated use of rotenone itself: Ball Pond, Connecticut was treated with 1.0 ppm synergized rotenone six time during a 17 year period to rid the 90 acre lake of golden shiner (*Notemigonus crysoleucas*). They always reappeared, and tolerance to the poison was suspected as at least part of the problem. Paired bioassays showed that the Ball Pond shiners were 4.0-7.1 times more tolerant than golden shiners from six ponds that had never been rotenoned.

Klingbeil (1975) discussed a related problem following rotenone treatment: any fish which remain in a lake that is not a "complete kill" may take advantage of the sudden reduction in competition for food and space, rapidly filling the ecological void left by the poisoned fish. The original survivors may be individuals that have a natural tolerance to rotenone (Tompkins, 1953; Meyer, 1966; Marking and Bill, 1976), indivdiuals that have an acquired tolerance (Oriciari, 1979), fish of an especially rotenone-resistant species, or simply individuals that found refuge from the poison in thick weeds, springs, etc. (Kiser et al., 1963; Prevost, 1960). Whatever the reason for their survival, there is the possibility that these fish may not only repopulate to former levels, but become an even greater nuisance than before poisoning. While this scenario differs in the strict sense from true acquired tolerance, the net result from a practical standpoint would be the same: more frequent and possibly higher-dosage treatments would be required to maintain a fishery.

Hubbs (1963) hypothesized that such a situation could occur in Texas waters, especially with partial rotenone treatments. Scholz (1983) believed that goldfish populations in eastern Washington lakes were increasing because rotenone treatments allowed surviving goldfish to expand into newly-vacant ecological niches. Whether or not this actually occurred is problematic, since there are no reliable fish population estimates for these lakes, and since the dates of first introduction are unknown. Even if such data were available, continued illegal stocking would tend to confuse any analysis unless there were some way to separate the descendants of freshly stocked fish from those of actual rotenone survivors.

Nuisance-fish increases have been documented in at least two lakes where rotenone treatments were unsuccessful: Jenkins (1956) reported that the carp population in Ardmore City Lake, Oklahoma exploded after a partial treatment. The goldfish population in California's Big Bear Lake likewise exploded following two unsuccessful treatments (Johnson, 1966; Hoover, CDF&G, pers. comm.). Klingbeil (1975) felt that the same thing might happen in Wisconsin lakes, and recommended restocking with gamefish as quickly as possible after poisoning to avoid such a takeover. He also suggested predator stocking.

On a statewide basis empirical evidence from Washington's 50-year history of fish poison use suggest that the above scenarios are not yet a problem: in the lakes that have been poisoned most frequently, the time between treatments has not decreased over the years.

Amphibians and Reptiles

Table Q lists toxicity data for amphibians. No laboratory data are available for reptiles. These tests suggest that larval amphibians such as tadpoles are far more susceptible to rotenone than metamorphosed adults. This stands to reason when we consider rotenone's high toxicity to gill-breathing forms.

The young of many amphibian species have completely metamorphosed and lost their gills by fall, when most rotenone treatment occurs. Others metamorphose during the fall, so that at least some individuals could be affected by rotenone treatment. In Washington, this category includes the spotted frog (*Rana pretiosa*), the red-legged frog (*Rana aurora*), the Northern leopard frog (*Rana pipiens*), the long-toed salamander (*Ambystoma macrodactylum*), and the roughskin newt (*Taricha granulosa*). Still others overwinter with gills: the Pacific giant salamander (*Dicamptodon ensatus*), the Cascades frog (*Rana cascadae*), and the bullfrog (*Rana catesbeiana*). The tiger salamander (*Ambystoma tigrinum*) never loses its gills, while the Northwestern salamander (*Ambystoma gracile*) is variable: some metamorphose in the fall, some overwinter with gills, and some retain gills for their entire life (Weschler, WDW, pers. comm). Larvae and gill-breathing adults of the above species could potentially suffer from routine fall rotenone treatments. Spring treatments could affect all species, since young amphibians are always in the gilled stage during that time of year.

Laboratory tests indicate that gill breathing amphibians have a relatively high tolerance to rotenone. Chandler and Marking (1982) reported that larval leopard frogs were 3-10 times more tolerant of rotenone than most of the 21 fish species tested by Markings and Bill (1976), and had about the same tolerance as the hardy goldfish. They noted that these animals were more sensitive to rotenone in the lab than in the natural environment, and concluded that they would probably be safe during lake treatments.

Denis and Devlin (1968) found that rotenone inhibited cell respiration and development in amphibian eggs. Lamy and Melton (1972) noted that rotenone produced unusual cleavage in leopard frog embryos. The laboratory procedures used in both these studies make extrapolation to the lake environment impossible. Again, however, frog and salamander eggs are not present in the fall when most rotenone treatments occur.

Table 9 Toxicity of rotenone to amphibians in laboratory bioassays.

Animal	Concentration (ppm)	Exposure	Formulation	Water Chemistry	Comments	Reference
Southern leopard frog larvae (Rana sphenocephala)	0.5	96 hr.	Noxfish	16° C; see Lennon & Walker (1964) for test conditions	LC50	Chandler & Marking 1982
Leopard frog (Rana pipiens)	7.3	24 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness	LC50	Farringer 1972
	7.9	24 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
	4.6	96 hr.	Dri-Noxfish	12° C, pH 7.2-7.6, 40-48 mg/l hardness		
	3.2	96 hr.	Dri-Noxfish	12° C, pH 7.6-8.0, 160-180 mg/l hardness		
Leopard frog tadpoles (Rana pipiens)	0.1	8-24 hr.	5% rotenone	---	100% mortality	Hamilton 1941
tiger salamander, with gills (Ambystoma tigrinum)	0.017	8-24 hr.	5% rotenone	---	toxic but not necessarily fatal	
tiger salamander, metamorphosed (Ambystoma tigrinum)	0.1	8-24 hr.	5% rotenone	---	100% mortality	
frogs	4.0 mg/kg body weight	---	pure rotenone	---	oral LD50	Haag 1931

Actual field data involving amphibians and reptiles are scarce and qualitative. When Brown and Ball (1943a) applied 0.5% ppm rotenone dust to a Michigan lake in early May, tadpoles were "greatly affected". Three months later, however, tadpoles were "extremely numerous", and the authors attribute it to post-rotenone breeding and the lack of predation by fish. High concentrations (~10 ppm) of Noxfish applied to ponds in Florida made alligators visibly ill, forcing them to leave the water (Fletcher, WDW pers. comm.).

In other field applications, Meehean (1942) noted that numerous salamanders (*Pseudobranchus striatus*) were killed by 0.5 ppm derris in five Florida lakes. The same author reported that 1.0 ppm derris killed the soft-shelled turtle (*Amyda ferox*).

Both adult and larval amphibians, as well as reptiles, may be indirectly affected by rotenone treatment. Most of Washington state's riparian herpetiles include fish and/or aquatic insects in their diets (Hodge, 1983; Stebbins, 1966), though none depend exclusively on these items. Aquatic insect reduction due to rotenone is rarely more than 71% in studied waters, and full recovery usually occurs within a month or two. Alternative food sources can probably support these animals during post-rotenone shortage of fish and benthos (State of California, 1983).

Birds

Oral toxicity for birds is listed in Table R.

The chipping sparrow is the most susceptible of the birds tested, with an LC50 of 113 mg pure rotenone per kg body weight. A six ounce chipping sparrow would require 19.2 mg pure rotenone, or 384 mg of the 5% fish-killing dust for a lethal dose. Similar calculations based on Brooks' (1961) work show that the lethal dose for a 6 ounce white rock chicken would be 1.02 ml Noxfish.

There would be no direct toxic effect of rotenone on birds and although no chronic, long-term toxicity studies have been performed on birds, the quick breakdown of rotenone and infrequent treatment of lakes and streams would decrease the likelihood of such effects.

As with mammals, only those birds which depend on fish or benthos for food such as: bald eagles (*Haliaeetus leucocephalus*), ospreys (*Pandion haliaetus*), loons (*Gavia spp*), kingfishers (*Megaceryle alcyon*), rails, grebes, and diving ducks - notably mergansers, buffleheads (*Bucephala albeola*), and goldeneyes (*Bucephala spp*) - could be affected indirectly by rotenone treatment of a lake. Except for the kingfisher, all these birds normally forage as adults over many miles and would probably not be harmed by the temporary loss in fish or benthic food following rotenone (Leschner, WDW, pers. comm.; State of California, 1983).

Table R Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to birds.

Animal	LD50	Formulation	Reference
White rock chickens	6 ml/kg	Chem-Fish Regular Noxfish	Brooks 1961
	8 ml/kg	Chem-Fish Special Pro-Noxfish	
Chickens (4-week)	>270 mg/kg	pure rotenone	Cutkomp 1943b
Chickens (5-day)	996 mg/kg	pure rotenone	Cutkomp 1943a
Chickens (5-day)	247 mg/kg	derris extract (25% rotenone)	
Eastern chipping sparrow (nestling)	113 mg/kg	pure rotenone	
Eastern song sparrows (nestlings)	130 mg/kg	pure rotenone	
Eastern robins (nestlings)	195 mg/kg	pure rotenone	
English sparrows (nestlings)	199 mg/kg	pure rotenone	
English sparrows (adults	853 mg/kg	pure rotenone	
pheasants (5-day)	850 mg/kg	pure rotenone	
pheasants (4-week)	1190 mg/kg	pure rotenone	
pheasants (3-4 month)	>1414 mg/kg	pure rotenone	Tucker & Crabtree 1970
prairie horned larks (adult)	450-500 mg/kg	pure rotenone	Cutkomp 1943a
mallards (3-4 month)	>2000 mg/kg	pure rotenone	Tucker & Crabtree 1970

Ospreys leave the Pacific Northwest beginning in September, returning in April, and thus would not be present during most treatments.

Kingfishers are highly territorial, so that the temporary disappearance of fish could force them off a lake and into competition with birds on other waters (Weschler, WDW, pers. comm.). Ducklings on a spring-rotenoned lake would be unable to forage on other waters, and may suffer reduced growth as an indirect result of rotenone treatment.

Mammals

Data on the acute toxicity of orally administered rotenone to mammals are listed in Table S. Only oral LD50's using aqueous solutions are shown, since these mirror the "real-life" situation. Schnick (1974) also conducted studies involving IP, IV, and IM injections of rotenone, as well as oral doses using unusual solvents.

The lowest LD50 of pure rotenone found in the literature on mammals is 55 mg/kg body weight for guinea pigs (Cutkomp, 1943b). To kill a small mammal weighing approximately half a pound would therefore require 12.5 mg pure rotenone, or 250 mg of the commonly used 5% dust. The smallest mammalian LD50 of rotenone formulation found in the literature is 170 mg/kg body weight of cube' powder (4.7% rotenone) reported by Haag and Taliaferro (1940) using male rats.

To produce subacute effects such as weight loss or liver damage also requires very high dosages fed continuously in the diet for many months. Rotenone is not likely to have a direct toxic effect on mammals in either the short or the long run. The reasons for the high mammalian tolerance to rotenone were discussed in the section describing the History of Rotenone. The EPA (1981) considers it safe to water livestock with rotenone-treated water.

Indirect effects might occur when rotenone disrupts the food supply for small mammals that feed on fish or benthos. In Washington this category includes mink (*Mustela vison*), river otter (*Lutra canadensis*), and water shrew (*Sorex palustris*).

Mink feed primarily on small mammals, with fish a secondary food source (Banfield, 1974). Additionally, they move frequently, all dens being temporary (Whitaker, 1980). River otters rely almost entirely on fish for food, and the temporary loss of prey following rotenone treatment may disturb them. But otters forage widely, sometimes travelling 50-60 miles during a year (Banfield, 1974), and would may not be displaced permanently. Water shrews may be indirectly affected by the temporary reduction in benthos (Weschler, WDW, pers. comm.).

Table 5 Median lethal dosages (LD50) of pure rotenone and rotenone formulations administered orally to animals.

Animal	LD50	Formulation	Reference
Rabbits	1.7 ml/kg	Chem-Fish Special Pro-Noxfish	Brooks 1961
White mice	350 mg/kg	pure rotenone	Kenaga and Allison 1971
Rats	1.5±0.1 ml/kg	Pro-Noxfish	Brooks 1961
	170 mg/kg	cubé (4.7% rotenone) in aqueous solution	Haag & Taliaferro 1940
	132 mg/kg	pure crystalline rotenone	Lehman 1951
	1500 mg/kg	derris	Lehman 1951
	1.5 cc/kg	Chem-Fish Special	Blue Spruce Co. 1973
Guinea pigs	60 mg/kg	pure rotenone	Cohen et al. 1960
	55-60 mg/kg	pure rotenone	Cutkomp 1943b

Human Health

Paths of Human Exposure to Rotenone - Figure 27 shows the uses of rotenone and how people may be exposed to it.

Direct contact with the dust used in fish control is a hazard faced mostly by fish biologists or other persons directly involved in the application. The ways in which the public could be exposed to the rotenone used in fish control are:

1) by eating fish killed with rotenone; or
2) by drinking water contaminated with rotenone.

Cohen et al. (1960) stated that the danger of ingesting rotenone by eating fish from poisoned lakes was very slight, since no significant amount would enter the fleshy part of the fish.

More recently, these residues have been quantified: following exposure to 2 ppm Noxfish, dead channel catfish, largemouth bass, bluegills, and redear sunfish contained from 0.045 to 0.101 ppm pure rotenone in their muscle fillets. Black bullheads which survived 1 ppm Noxfish for one hour contained 0.05 ppm pure rotenone immediately following treatment, and less than 0.020 ppm pure rotenone after 12 hours in fresh water (State of California, 1985). Based on the maximum residue figure and an estimated lethal dose of 18 g pure rotenone, researchers stated that a 130-pound person would have to eat a minimum of 397 pounds of fish at once to receive a lethal dose.

The California Department of Health Services suggested an acceptable daily intake (ADI) for humans of 0.0004 mg pure rotenone/kg body weight/day, applying a safety factor of 1,000 to the 0.4 mg/kg/day no-observable-effect levels (NOEL) determined by the Midwest Research Institute (1980). A 130-pound person would have to eat daily about one-half pound of fish containing 0.100 ppm pure rotenone to reach the ADI, not allowing for probable losses of rotenone through natural degradation and cooking (State of California, 1985). Canada allows a residue of 0.1 ppm pure rotenone in food (Khera et al., 1982).

The original use of rotenone-bearing plants in South America was the collection of fish for the table (Teixeira et al., 1984; Moretti and Grenand, 1982).

The main path by which people may come into contact with rotenone from fish applications is through drinking water (Gosa'lvez and Di'az-Gil, 1978). Cohen et al. (1960) concluded that the use of rotenone to kill fish in public reservoirs was consistent with the objective of safe and potable water. Where natural processes did not thoroughly detoxify rotenone by the time it reached the treatment station, they suggested the use of activated carbon to remove the residue.

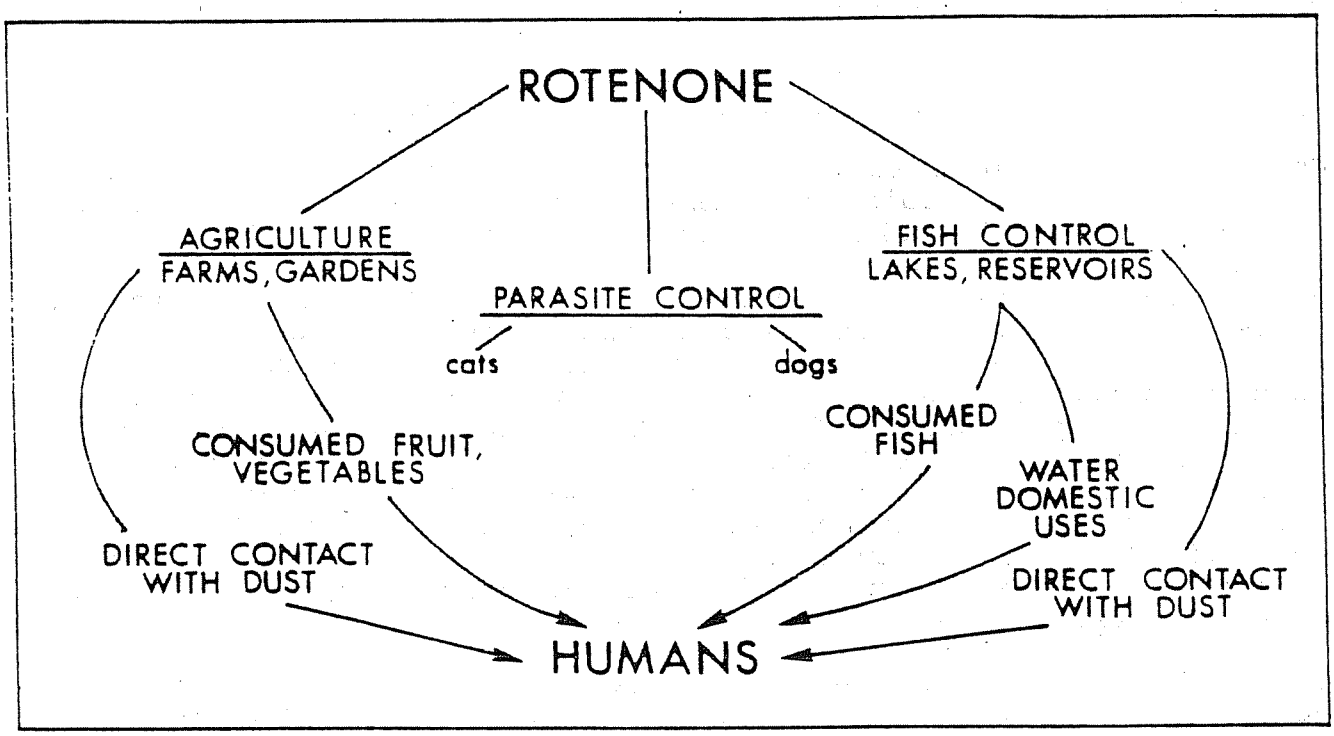

Figure 27 Paths of possible human exposure to rotenone. Source: Gosálvez and Díaz-Gil 1978.

Acute Oral Toxicity - There has never been a human death attributed to rotenone (Gosselin et al., 1984; Schnick, 1974; Thienes and Haley, 1972). The lethal oral dose for humans has been estimated from laboratory test with other mammals, mostly rats, and these estimates are shown in Table T. Lethal doses for pure rotenone range from 0.1 g per kg of body weight to 1.4 g/kg.

Santi and Toth (1965) warned that, contrary to most current literature, rotenone could be highly toxic to humans. Their experiments gave oral LD50's for rats that went as low as 0.049 g/kg, which is half of the smallest lethal dose listed in Table T. Yet their experimental solvent was acetone, and Santi and Toth admitted that to "render this (high) toxicity evident, it is necessary to choose proper solvents (ethanol and acetone) . . ."

Finally, while no record exists of a human fatality due to rotenone, there are several anecdotal reports of deaths due to the plants from which rotenone is extracted: Moretti and Grenand (1982) mention the use of *Lonchocarpus* by natives in French Guiana to commit suicide; Gimlette (1929) cites the use of "tuba root" *Derris elliptica* in Malaya for abortions, ritual suicide, and even attempted murder; Campbell (1916) describes a suicide in Singapore due to oral ingestion of *D. elliptica*. None of these references make mention of the dosages, and fresh derris root has a much higher toxicity than the dried powdered root from which rotenone is extracted (Gosselin et al., 1984).

Acute Respiratory Toxicity - Rotenone is more toxic when inhaled than when eaten (Windholz, 1983; Ambrose and Haag, 1937), though no estimates of the lethal respiratory dose for humans have been published. Santi and Toth (1965) tested a spray-mist of pure rotenone and ethanol on rats, and concluded that when inhaled "in proper vehicles or in association to other drugs, rotenone might cause unpleasant surprises". The "proper vehicle" they refer to would be a solvent such as ethanol or acetone. It is therefore highly unlikely that acute respiratory poisoning could occur in routine fisheries work.

Symptoms of Acute Rotenone Poisoning - Symptoms of acute oral rotenone poisoning are largely inferred from animal studies. Onset of the symptoms occurs within minutes to 5-6 hours after first coming in contact with the poison (Lehman, 1951). Poisoning results in numbness of the oral mucous membranes, nausea, vomiting, gastric pains, and muscle tremors. Respiration is at first stimulated, then depressed. Convulsions and coma are followed by death. The immediate cause of death is ashpyxia from repiratory arrest (Gosselin et al., 1984; Thienes and Haley, 1972; Windholz,

1983; Sax, 1984). Symptoms of acute respiratory poisoning are the same except that there is also some lung irritation (Gosselin et al., 1984).

Table 7 Estimated lethal oral doses of rotenone for humans.

Lethal oral dose of pure rotenone (g/kg body weight)	Reference	Lethal oral dose of pure rotenone for a 150 lb. human	Lethal oral dose of 5% rotenone formulation for a 150 lb. human
0.3 - 0.5 g/kg	Gosselin 1984	20 - 34 g	400 - 680 g (14 - 24 oz)
0.3 - 0.4 g/kg*	Arena 1979	20 - 30 g	400 - 600 g (14 - 21 oz)
0.132 g/kg	Dreisbach 1983	9 g	180 g (6 oz)
0.14 - 1.4 g/kg**	Arena 1979	10 - 100 g	200 - 2000 g (7 - 70 oz)
0.20 g/kg	Sax 1984	14 g	280 g (10 oz)
---	Deichmann & Gerarde 1969	10 g***	200 g (7 oz)
0.132 g/kg	Lehman 1951	9 g	180 g (6 oz)
0.100 - 0.199 g/kg	U.S. EPA 1970	7 - 14 g	140 - 280 g (5 - 10 oz)

* actually reported as "20 - 30 g for a 150 - lb. man."

** actually reported as "10 - 100 g/70 kg."

*** actually reported as "the probable lethal dose (oral) for an adult."

Although rotenone has the potential to be highly toxic to humans when combined with certain solvents (Santi and Toth, 1965), there are certain properties of rotenone used in fisheries applications that reduce this potential:

1) the low percentage (1 to 5%) commonly used in commercial prepartations (Gosselin et al., 1984);
2) it has an extremely low solubility in water (Santi and Toth, 1965);
3) it is unstable in nature, detoxifying quickly in both light and air (Haley, 1978);
4) it is an irritant when eaten, causing prompt vomiting (Haag, 1931);
5) it is inefficiently absorped in the gastrointestinal tract (Gosselin et al, 1984);
6) the human body contains an effective oxidizing enzyme system (Schnick, 1974; Haag, 1931; Santi and Toth, 1965).

Subacute Toxicity - Direct Contact - WDW fisheries biologists handling rotenone dust during the course of routine lake poisonings usually report one or more of the following symptoms: a numb sensation in the mouth and lips, a mild sore throat, mild headache, eye irritation, and a runny nose (pers. comm.). Fisheries biologists in California, exposed to rotenone dust more or less continuously for periods up to three weeks developed all the above symptoms as well as sores on mucous membranes, eczema-like rashes, sloughing of the skin in some areas, severe week-long eye inflammations, and loss of appetite and the ability to taste (Pintler and Johnson, 1958).

Exposure to derris powder resulted in violent dermatitis of the genital region, irritation of the tongue and lips, and nasal passage inflammation (Racouchot, 1939). Both these studies recommended the use of face masks or protective clothing to reduce symptoms.

There has been no long-term study on the subacute effects of direct contact with rotenone dusts or liquids. The U.S. Environmental Protection Agency (1981) considers it safe to swim in water treated with rotenone. Dawson (1991) concluded that based on low mammalian toxicity and rapid rate of decomposition (especially at warmer temperatures that might be appropriate for swimming), the margin of safety is so great that water would be safe for swimming and other recreational use immediately following treatment.

Subacute Oral Toxicity - As with acute poisoning, the long-term toxic effects of rotenone on man must be inferred from experiments on other mammals. Table U presents the results of long-term oral dosages of rotenone on rats, dogs, and hamsters.

As shown in Table U, the most commonly noted effects of long-term rotenone feeding were:

1) Liver changes . Where noted, this usually involved a fatty metamorphosis of the liver. the lowest dosage that ever produce these changes was a continuous diet of food containing 130 ppm derris powder (9.6% rotenone) for 190 days. The same authors found no liver changes in dogs fed three times that amount for 240 days. No investigators since 1942 have reported these liver changes, although close histological inspection of all internal organs was part of all the later studies.

2) Growth inhibition . Either a major or minor decrease in weight gain, when compare to control animals, was reported in 10 fo the 13 studies. In some cases this may be a result of the unpalatability of the rotenone formulation, but Haag (1931) fed his dogs rotenone in capsule form and Freudenthal et al. (1981) took care to make the hamster diet equally palatable for both test and control animals. In both these studies significant growth inhibition was reported.

3) Other effects . Midwest Research Institute (1980) found that dogs fed and 10 mg of pure rotenone per kg of body weight developed gastrointestinal problems. The high dose also caused mild anemia and small but consistent decreases in blood glucose, total lipids, and cholesterol.

Studies have also involved pregnant mammals to determine if oral doses of rotenone would affect the fetuses of newborns. Hazelton Raltech, Inc., (1983; 1982; 1981) conducted three studies with pregnant rats, and determined that rotenone neither killed fetuses nor produced abnormal young when fed to the mothers on days 6 through 19 of gestation at doses ranging form 0.75 to 15 mg pure rotenone/kg body weight/day. The 1983 study involved feedings of up to 75 ppm pure rotenone tot two successive generations of rats on a daily basis; there was no effect on reproductive performance of either sex. Khera et al. (1982), in a 9-day study with pregnant rats, found that daily oral doses of 5 and 10 mg pure rotenone/kg body weight were responsible for a higher rate of nonpregnancies and resorptions, while 2.5mg/kg had no effect on the mothers or the young. Freudenthal et al. (1981) noted that a continuous diet of 500 ppm pure rotenone fed to a pregnant hamster for three months was toxic to the embryos and resulted in cannibalism of the young by the mothers.

Table 5 Results of long-term oral dosages of rotenone on dogs, rats, and hamsters.

Test animal	Period of daily treatment	Daily dosage, formulation	Liver change	Major growth inhibition	Minor growth inhibition	Other changes	No effect	Reference
dogs	102 days	10 mg (pure rotenone)	x	x				Haag 1931
dogs	180 days	0.4 mg/kg					x	Midwest Research Institute 1980
		2.0 mg/kg			x	x		
		10.0 mg/kg (pure rotenone)				x		
dogs	240 days	400 ppm (derris powder, 9.6% rotenone)			x			Ambrose and Haag 1938
dogs	190 days	130 ppm (derris powder, 9.6% rotenone)	x					Ambrose and Haag 1938
dogs	840 days	50 ppm					x	Hansen et al. 1965
		150 ppm					x	
		400 ppm (cubé powder, 5.8% rotenone)					x	
rats	150 days	156 ppm	x					Ambrose and Haag 1938
		312 ppm	x		x			
		625 ppm (derris powder, 9.6% rotenone)	x	x				
rats	150 days	10 ppm					x	Haag and Taliaferro 1940
		25 ppm					x	
		75 ppm					x	
		150 ppm					x	
		300 ppm (cubè powder, 4.7% rotenone)					x	

Table U Continued

Test animal	Period of daily treatment	Daily dosage, formulation	Liver change	Major growth inhibition	Minor growth inhibition	Other changes	No effect	Reference
rats	200 days	600 ppm 900 ppm 1200 ppm (derris powder, 0.6-9.6% rotenone	x x x	x x x				Ambrose et al. 1942
rats	200 days	1200 ppm (cubé powder, 2.9% rotenone)			x			Ambrose et al. 1942
rats	730 days	50 ppm 100 ppm 250 ppm 500 ppm 1000 ppm (cubé powder, 5.8% rotenone)			 x x x x		x	Hansen et al. 1965
rats	490 days	100 ppm (Pro-Noxfish)		x				Brooks and Price 1961
rats	365 days	100 ppm (detoxified Pro-Noxfish)			x			Brooks and Price 1961
hamsters	90 days	500 ppm 1000 ppm (pure rotenone)		 x		x x		Freudenthal et al. 1981

Carcinogenicity - The results of a number of studies on the long-term effects of rotenone dusts (between 0.6 and 9.6% pure rotenone) were published from 1931 to 1942 (Haag, 1931; Ambrose and Haag, 1938; Haag and Taliaferro, 1940; Ambrose et al., 1942; Ambrose and Haag, 1936). While their results varied (see Table V), no tumors were observed by any of the researchers.

The first mention of tumors possibly caused by rotenone appeared in Lehman, 1952. He reported an increased incidence of peculiar cell masses - classified between hyperplasia and tumor - in the livers of rats fed rotenone continuously. These growths appeared in the rats fed between 2 and 10 ppm pure rotenone in the diet, but not at higher levels.

In 1959, another study by the U.S. Food and Drug Administration concluded that there was an abnormal incidence of liver tumors in rats fed 2, 5, and 10 ppm pure rotenone in the diet for two years. These tumors did not appear at higher levels (unpublished internal report, U.S. FDA, Division of Pharmacology, 1959; reported by Gosalvez, 1983).

Studies since that time on the cancer-causing potential of long-term exposure to rotenone are shown in Table V. All except two studies used pure rotenone in the tests; Hansen et al. (1965) fed cube' powder with 5.80% rotenone, similar to the commercial dusts used for fish control, and Brooks and Price (1961) fed Pro-Noxfish. In addition to the fresh powder, these last authors also tested Pro-Noxfish that had been completely detoxified, to see if the residues left in the water had any long-term effects.

Gosalvez and Merchan (1973) published a study in which rats injected with rotenone developed mammary tumors (Table V). Although these tumors were benign, they were transplantable, and showed an average doubling time of 2-3 months. The tumors were in many ways morphologically similar to human breast cancer (Gosalvez et al., 1977).

The same authors reported that these tumors could also be produced by low-level oral doses of rotenone on a daily basis for 45 days. They suggested a possible hormonal mechanism for the inducement of the tumors caused by rotenone (Gosalvez et al, 1979), and warned that rotenone could be "reaching the human female in certain countries" in amounts sufficient to cause mammary tumors, mostly by way of garden vegetables and drinking water (Gosalvez and Diaz-Gil, 1978).

As a result of this research, the U.S. EPA scheduled a reevaluation of rotenone and placed it on the Rebuttal Presumption Against Registration listing (RPAR) in 1976 (Anon, 1976). The agency commissioned a three year study, the results of which are shown in Table V (Freudenthal et al., 1981). The researchers concluded that neither direct oral administration, inclusion in the

Table V Studies on the cancer-causing potential of long-term exposure to rotenone.

	Animal	Dosage	Vehicle	Diet	Fed or treated for	Observed for	Tumors	Reference
ROTENONE FED IN DIET OR WATER	Syrian golden hamsters	125, 250, 500, & 1000 ppm (pure rotenone)	diet	Purina hamster chow	18 months (daily)	18 months	NO	Freudenthal et al. 1981
	mice (two lab strains)	3 ppm (pure rotenone)	diet	not reported	18 months (daily)	18 months		Innes et al. 1969
	Osborne-Mendel rats	50, 100, 250, 500 & 1000 ppm (cubé powder, 5.80% rotenone)	diet	Purina laboratory chow	24 months (daily)	24 months	NO	Hansen et al. 1965
	Carworth rats	100 ppm (Pro-Noxfish)	drinking water	Gaines dog meal	17.5 months (daily)	17.5 months	NO	Brooks and Price 1961
	Carworth rats	100 ppm (detoxified Pro-Noxfish)	drinking water	Gaines dog meal	13 months (daily)	13 months	NO	Brooks and Price 1961
ROTENONE FORCE-FED	Wistar rats	2-3 mg/kg (pure rotenone)	sunflower oil	"deficient diet"	60 days (daily)	14 months	YES	Gosálvez et al. 1977
	Wistar rats	1.7 & 3.0 mg/kg (pure rotenone)	Mazola corn oil	Purina rat chow	42 days (daily)	14 months	NO	Freudenthal et al. 1981
ROTENONE INJECTED	Sprague-Dawley	1.7 & 3.0 mg/kg (pure rotenone)	Mazola corn oil	Purina rat chow	42 days (daily)	17 months	NO	Freudenthal et al. 1981
	albino rats	1.7 mg/kg (pure rotenone)	sunflower oil	"deficient diet"	42 days (daily)	18 months	YES	Gosálvez and Merchán 1973
	Wistar rats	9.0 mg total dose (pure rotenone)	---	"enriched" rat diet	---	18 months	NO	Gosálvez 1983

diet, or IP injection of rotenone caused tumors. As a result, the EPA dropped rotenone from its RPAR list in 1981 (Anon., 1981; 1983).

Marking (1988) also performed studies on chronic oral toxicity in rats, effects on reproduction in rats, and subchronic oral toxicity in dogs and concluded from the results of these studies and those in the literature that even high doses of rotenone do not cause tumors or reproductive failure, nor adversely affect fetal development.

APPENDIX A

FORMS

PRE-REHABILITATION PLAN

I. PROPOSAL

A. Justification for Proposed Rehabilitation

1. Demonstrate declines in the catch, survival, and/or size of trout fry (or other game fish).
2. Estimate number of recreational days lost due to poor trout fry (or other game fish) survival.
3. Demonstrate declines in past waterfowl use (if applicable).
4. For new waters, demonstrate the water's potential to produce a viable game fish fishery.

B. Physical Description of the Water Proposed for Rehabilitation

Provide the best map available with the following details:

1. Name of water (and county).
2. Location using township coordinates of proposed water.
3. Surface acres of water.
4. Depth range (and contours if available). If the water is greater than 100 acres, a bathymetric map will be produced if not available.
5. Volume of water.
6. Outlet statistics - Permanent, intermittent, dry.
7. Stream miles, stream flow.
8. Number of developed public access areas.
9. Land ownership (%) Public ___ Private ___
10. Established resorts.

C. Proposed Management Actions

1. Target species.
2. Date of last rehabilitation.
3. Proposed treatment date.
4. Estimated restocking date.
5. Species to restock.
6. Number of catchables, fry to stock.
7. Proposed toxicant name, type (liquid or powder) concentration, and amount required.
8. Method of application.
9. Size of crew and number and name of crew leaders needed.

II. PURPOSE

Detail the purpose of the rehabilitation and how this action relates to the management plan for this water.

III. INTENDED OUTCOME\MEASURE OF SUCCESS

Estimate duration of beneficial effects and how this will be measured.

IV. RESOURCE IMPACTS

1. Detail potential impacts to non-targeted resources, using survey data of individual waters (including outlets), information from non-game and waterfowl programs, and documented levels of impacts from published studies (use Bradbury for references).
2. Detail potential impacts to human related uses of the water or shoreline (i.e. irrigation, drinking water, beach combing, temporary loss of fishing, etc.) Identify the existence of water intakes.
3. List any endemic species, and/or species which are rare, endangered, threatened or otherwise listed which may be impacted by the proposed rehabilitation.

V. MITIGATING FOR ADVERSE IMPACTS

1. Describe how adverse impacts can be mitigated, or softened (i.e. time of year, removal of dead fish from shoreline, etc.)
2. Describe measures to protect downstream resources (list detoxicant used if applicable).
3. Describe measures to protect endemic species, and/or species which are rare, endangered, threatened and/or otherwise listed which may be impacted by the proposed rehabilitation.
4. Describe the safety precautions for pesticide applicators which will prevent health hazards.
5. Describe how the public will be discouraged from collecting dead or dying fish.

VI. RECREATIONAL IMPACT

Estimate increased angler success and number of recreational days generated from the proposed rehabilitation.

VII. ECONOMIC IMPACT

Given the above increased days in recreation, estimate impact to local businesses, and costs and benefits to our program. (Use Bradbury 1986 for reference).

VIII. RELATED MANAGEMENT ACTION

Detail management actions which are related to the proposed rehabilitation (e.g. stocking sizes and levels of fish, pre-rehab removal of selected fish, etc.)

IX. PUBLIC CONTACT

Detail how and when the public was contacted and what was the public's general response to the proposal.

POST REHABILITATION FORM

1. Lake or Stream _____ County _____
   Section _____ Township _____ Range _____, WM

2. Lakes - surface acres____ Miles of inlet or outlet treated____

3. Steams - miles treated ____ Miles of tributaries treated _____

4. Maximum depth _____ Average depth _____

5. Weight (lbs) of water treated _____ Toxicant used_____

6. Amount used _____lbs.; _____% active ingredient
   _____gals.; _____% active ingredient

7. Concentration applied _____ppm, Date treated _____

8. Man hours expended in preparation, treatment and cleanup _____
   Air time used _____

9. Conditions in the lake on date of treatment:

Depth in feet	**Temperature**	**pH**	**Dissolved Oxygen**
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

10. Species of fish eradicated in order of relative abundance:
    1. _____ 5. _____
    2. _____ 6. _____
    3. _____ 7. _____
    4. _____ 8. _____

11. Possibility of a complete kill: _____

12. Detoxicant used _____
If any, report on effects recorded on downstream fishery.

13. Period of toxicity: _____

14. Description of treatment and other comments:_____
_____
_____
_____
_____
_____
_____
_____
_____

_____ _____
Fishery Biologist Date
Region _____

APPENDIX B

HISTORY OF ROTENONE

HISTORY OF ROTENONE

Rotenone is a white crystalline ketone with the chemical formula $C_{23}H_{22}O_6$. It is found in the roots of several tropical plants grown in Malaya, the East Indies, and Central and South America. For centuries, natives in these areas have killed fish for the table by poisoning lakes, ponds, and streams with rotenone preparations.

Besides rotenone itself, the so-called fish poison plants contain other active ingredients called rotenoids which are chemically related, be generally less toxic.

Rotenone and its parent plants have hundreds of common names, but the most widespread are derris, tuba (both names used to describe Asian genus *Derris*, especially *D. eliptica*), timbo', cube', and barbasco (the last three referring to the South American genus *Lonchocarpus*, especially *L. utilis, L. urucu,* and *L. nicou*).

Rotenone is used primarily as an agricultural insecticide and in household gardens. Its use as a fisheries management tool began in 1934, when Dr. Carl Hubbs attempted to poison carp and goldfish in two small Michigan ponds. By 1970, all states except Hawaii had used rotenone to kill fish, and most were using it routinely.

By far the most common aquatic use of rotenone today is the improvement of sport fishing via the elimination of other non-game or competitor species. Some other aquatic applications in the U.S. and Canada have been reported in the literature: Weier and Starr (1950) improved waterfowl refuges by poisoning carp from pond where they had uprooted the natural vegetation used by ducks for food and shelter; rotenone has been used to sample fish populations in lakes (Krumholz, 1950a), streams (Boccardy and Cooper, 1963), and coral reefs (Smith, 1973); M'Gonigle and Smith (1938) used rotenone to create a disease-free water source for a hatchery; and municipal water supplies have been treated with rotenone to reduce turbidity and algae caused by bottom-feeding fish (Hoffman and Payette, 1956; Bonn and Holbert, 1961; Barry, 1967).

The United States which consumes about 15 million pounds of rotenone per year, is supplied mostly by South America. Commercial preparations used in agriculture and fisheries are made primarily from the resins and dried and ground roots of *Derris* (and Asian genus) and *Lonchocarpus* (a South American genus) which are cultivated for that purpose. These dusts therefore contain not only rotenone itself (usually about 5% of the total content) but also varying amounts of the other rotenoids, as well as biologically inert material.

Synergists are sometimes added. Pure rotenone for laboratory purposes is extracted from the resins with solvents such as chloroform and benzene.

Technical literature on the sources, chemistry, history and use of rotenone abounds. The preceding is only a brief summary from the following detailed sources: Haley, 1978; National Academy of Science, 1983 (literature reviews of rotenone's chemistry, extraction, toxicology, biotransformation, and carcinogenicity); Schnick, 1974 (exhaustive literature review on all fisheries uses); Lennon, et al., 1970 Eschmeyer, 1975 (fish toxicants in general, with numerous references to rotenone and its history in fisheries); Gosa'lvez and Di'az-Gil, 1978 (scope of commercial use); Moretti and Grenand, 1982; Teixeira, et al., 1984 (botany and use of fish poison plants).

How Rotenone Works

Regardless of the organism, rotenone's primary toxic action is at the cellular level, where it blocks oxidative phosphorylation (Fukami, et al., 1967; Lindahl and Oberg, 1961; Ernster, et al., 1963; Figueras and Gosa'lvez, 1973). the specific site of action is localized in the electron transport system, where it becomes tightly bound (Oberg, 1961; Horgan, et al., 1968). Teeter, et al. (1969) demonstrated that high concentrations of rotenone can inhibit electron transfer in more than one region of the respiratory chain. Both the lethal and numerous pharmacological effects of rotenone can be ascribed to its inhibitory effect on cellular metabolism (Santi and To'th, 1965).

Rotenone's ability to inhibit cellular respiration has been well documented in cells of mammals, fish and insects (e.g., Fukami, et al., 1970), as well as amphibians (Denis and Devlin, 1968), and even plants (Ikuma and Bonner, 1967). Why then is rotenone extremely toxic to some life forms (fish and insects), relatively nontoxic to others (mammals, including humans) and virtually nonphytotoxic (being used extensively on crops and garden plants)?

Fukami, et al. (1969,1970) concluded that the selective toxicity of rotenone between mammals, fish and insects was due to the differences in the site of entry and/or ease of rotenone detoxification rather than any cellular differences in the oxidation chain of these animals. There are some minor variations, however, in the mitochondria of different animals (and organs within a species) that may also contribute to these differences in toxicity (Ilivicky and Casida, 1969).

Although rotenone is toxic to isolated mammalian mitochondria, mammals - including humans - are not highly susceptible to the poison because they are protected by effective oxidizing enzyme systems (Shnick, 1974; Haag, 1931; Santi and To'th, 1965) and because of slow, inefficient gastrointestinal absorption (Gosselin, et al., 1984). If rotenone is enabled to reach its site of action through the use of solvents such as ethanol or acetone, however, there is no real difference in the sensitivity to the poison

between fishes and warm-blooded animals (Santi and To'th, 1965; Schmidt and Weber, 1975). Also while absorption in mammals is very inefficient, extremely high or continuous dosages may allow enough rotenone to reach the site of action for toxic effects to appear.

The high susceptibility of fish to rotenone is mostly due to its efficient entry through the gills (Schmidt and Weber, 1975; Oberg, 1964, 1967b). Oberg (1967a) demonstrated that the specialized structure of gills and lipid solubility favored the entrance of rotenone from water - where it is virtually insoluble - into the gill cell membrane. Once in the bloodstream rotenone is quickly carried to vital organs (such as the brain), where it inhibits cellular respiration (Oberg, 1964). The fact that fish immersed in rotenone solutions are protected if their gills are in contact with pure water is further proof that the gills are the main entry site in fish (Oberg, 1964). Orally administered rotenone does have a toxic effect on fish, but not nearly so much as topically applied rotenone (Hashimoto and Fukami, 1969).

Previously, rotenone was thought to kill fish by either destroying the gill tissues (Danneel, 1933) or by constricting the tiny gill capillaries (Hamilton, 1941). Microscopic examination of the gills of both fish and aquatic insects revealed that death usually occurred without any gill vasoconstriction or deterioration (Oberg, 1959; Lindahl and Oberg, 1961; Claffey and Ruck, 1967). However, gill epithelium may be damaged by high concentration of rotenone as a side effect, and when this occurs, the fish may die even when cellular respiration is restored by placing the fish in fresh, untreated water (Oberg, 1967b).

As in fish, the high susceptibility if insects to rotenone is primarily due to easy entry via the gill-like tracheae and the cuticle, although rotenone can also enter effectively through the mid-gut (Tischler, 1935; Fukami, et al., 1970).

In both aquatic insects and fish, rotenone tolerance tends to vary inversely with oxygen requirements, as would be expected for a poison that inhibits respiration (Engstrom-Heg, et al., 1978).

Rotenones toxic effects are reversible, depending on the amount absorbed by the animal. Natural detoxification of sublethal rotenone dosages in insects, fish and mammals is primarily via oxidation by microsomal mixed function oxidase (mfo) enzymes (Fukami, et al., 1969; Fabacher and Chambers, 1972; Ludke, et al., 1972). In fact, certain chemicals (such as Sesamex) known to inhibit these mfo enzymes are sometimes added to insecticidal rotenone preparations as a synergist to increase its toxicity. At least in mammals, the inhibitory effect of rotenone on mitochondria is overcome by adding vitamin K (menadione), which activates a bypass of the rotenone-sensitive site (Santi and To'th, 1965; Gosselin, et al., 1984).

In fish, these natural mechanisms are sometimes able to effectively counter rotenone poisoning if the fish is removed to fresh, untreated water. While Leonard (1939) and Brown and Ball (1943a) were unable to revive rotenone-poisoned fish that had lost their equilibrium, Smith (1940) found that brook trout recovered in a fresh water bath, even when rotenone had affected their ability to swim upright. Gilderhus (1972) performed laboratory tests demonstrating that fish which had been floating on their sides in lethal concentrations of rotenone for as long as four hours often recovered if they were placed in fresh, untreated water. Oberg (1967b) revived rotenone-poisoned cod in untreated water and suggested the metabolic pathways involved.

In addition to fresh water baths, biologists have apparently succeeded in reviving fish with at least two other techniques. Bouck and Ball (1965) revived a variety of warmwater fish in methylene blue solutions. They tried the stain after Oberg (1961) showed that it reduced respiratory inhibition due to rotenone in the mitochondria of rat livers and fish gills. In one of their tests, Bouck and Ball were able to show that neither fresh water alone nor very low concentrations of methylene blue revived fish. The technique was not effective on rainbow trout, and the authors also cautioned that the stain was toxic to higher aquatic plants and that it encouraged bacterial growth on fish.

Fletcher (1976) successfully revived rotenone-poisoned bass on four Washington state lakes using a potassium permanganate dip. These fish were then moved by hatchery trucks to other lakes where they were released. Many of the fish that later recovered showed no signs of life when initially placed in the hatchery trucks. Fletcher hypothesized that the 20-second permanganate dip worked by neutralizing residual rotenone on the gills and body surface of the fish. Hepworth and Mitchum (1966), who also revived fish with permanganate dips and fresh water, concurred that the chemical neutralized residual rotenone on the gills. Fletcher also suggested that the extremely cold, hyperoxygenated fresh water in the hatchery trucks aided recovery. But since all fish in both Flethcer's and Hepworth and Mitchum's tests received the dip, there is no way to tell which factor was responsible for the recovery. It is possible that the cold, oxygenated fresh water alone would have revived the fish. Bouck and Ball (1965) stated that while permanganate detoxified rotenone in water, it was of no value in reviving fish..

Rotenone is unstable, degrading rapidly with exposure to light, heat, oxygen and alkalinity (Lennon, et al., 1970; Schnick, 1974). The degradation products were originally identified as dehydrorotenone (which is non-toxic to fish) and water (Subba-Rao and Pollard, 1951). Cheng, et al. (1972) later identified 20 degradation products, mainly rotenoids.

In natural waters, a variety of other factors contributes to the rate of degradation. These include the presence of organic debris, turbidity, lake morphology, dilution by inlets and runoff, and the dosage used (Shnick, 1974).

Post (1958) was the first to quantify the rate of rotenone detoxification in water. He concluded that water temperature was the most significant factor in the breakdown of rotenone; total dissolved solids, pH, alkalinity, dissolved oxygen, and various other cations and anions did not change the rate of breakdown to any great extent, and were not useful as predictive tools. He derived two empirical equations based on temperature for determining the time to detoxification.

More recent field and laboratory research has shown deviations from Post's predictive equations; these turned out to be related to the amount of sunlight reaching the toxic water. As noted above, rotenone is photochemically unstable, degrading rapidly in sunlight, and this reaction is accelerated at higher temperatures. Rotenone was shown to detoxify quickly in shallow warm lakes and slowly in deep or ice-covered lakes (Meyer, 1966; Engstrom-Heg and Colesante, 1979). The darker waters of the hypoliminion also detoxify more slowly than the well-lit epilimnetic water in a given lake (Engstrom-Heg and Colesante, 1979).

With these additional factors in mind Engstrom-Heg and Colesante (1979) developed the most complete set of equations for predicting rotenone breakdown in a wide variety of lakes and ponds. Their results in epilimnetic waters coincided closely with Post's (1958) earlier findings, and two of their predictive equations for use in clear, shallow, unstratified ponds are simple modifications of Post's formulas. But they added that the reduced sunlight in the hypolimnetic waters played an important role in the slow breakdown of rotenone in other lakes, and they developed three additional equations that take this into account. These equations are practical for field use, requiring only standard limnological data that area already available for most lakes. Engstrom-Heg and Colesante's detoxification rates coincided very closely with the results of Markings and Bills (1976), who arrived at their rate constants using a totally different approach.

While toxic periods vary greatly depending on the factors mentioned above, most lakes treated with rotenone are completely detoxified within five weeks of treatment (Shnick, 1974). Lakes in Washington state are usually non-toxic to fish about four to five weeks after treatment.

It is possible to accelerate the natural breakdown of rotenone in water by using certain oxidizing chemicals such as chlorine or potassium permanganate (Dawson, 1975). Considering the high rate of natural rotenone breakdown and the quantity of water involved,

these chemicals have little practical value in lakes. No lake detoxification with chemicals has been recorded in the literature (Lennon, et al., 1970).

Potassium permanganate is sometimes used, however, to detoxify outlet streams that flow from treated lakes (Engstrom-Heg, 1972). Pfeifer (1985) describes its use in detail and cites two case histories in western Washington. Both chlorine and activiated carbon have been used to detoxify and deodorize treated lake water as it entered municipal water supplies (Cohen, et al., 1960; 1961a; 1961b).

Commercial fish-killing preparations of rotenone fall into three basic categories (Schnick. 1974):

1) 5% powder;
2) 5% emulsifiable concentrate;
3) 2.5% synergized emulsifiable concentrate.

Emulsifiable concentrates were developed to make application easier and to aid in dispersing the product (Meyer, 1966). Synergists (usually organic solvents such as sulfoxide) were later added to some formulations. These synergists aid absorption of the poison so that a 2.5% synergized mixture can be as effective as the more costly mixtures containing 5.0% rotenone (Price and Calsetta, 1957). Marking and Bills (1976) made extensive laboratory tests and found that the 25% synergized formulation Pro-Noxfish somewhat more toxic than a 5.0% nonsynergized formulation (Noxfish) to rainbow trout. the synergist sulfoxide, the emulsifying agents, and the solvents used in these preparations have been tested and found innocuous themselves (Penick and Co., 1959).

Bassett (1956) tested to see if there were significant toxicity differences between 2.5% preparations (Pro-Noxfish and Chem-Fish Special) and a 5% preparation (Chem-Fish); he found that in terms of toxicity, they were basically the same. Shannon (1969) tested nine commercial formulations ranging from 2.5% to 7.5% rotenone content. His laboratory bioassays with sunfish showed little variation in the amounts of formulation needed to produce a 24-hour LC50; he concluded that cost, mixing ability, and ease of handling should therefore determine the formulation used. Marking and Bills' (1976) laboratory tests showed no significant difference in toxicity between Noxfish and 5% rotenone powder. (Commercial preparations mentioned by trade name are shown in Table B.)

Although there are some conflicting reports, most investigators reported that rotenone was more toxic at high than at low temperatures, in acid than in alkaline waters, and in soft than in hard water. Many of these were field studies, however, where a great many other unmeasured variables could have affected the results. Furthermore, efficacy in many of the early laboratory and

caged-organism studies was based on survival time of the test organism rather than on concentration of the toxicant (Marking and Bills, 1976).

In the most recent, extensive and statistically thorough research on this topic, Marking and Bills (1976) found only slight changes in the toxicity of rotenone at differing temperature (44-72$_{o}$F), pH (6.5-9.5), and water hardness (10-300 mg/l CaCO). These test were performed under standardized laboratory conditions using rainbow trout, channel catfish, and bluegills.

Burdick, et al. (1955) also concluded from bioassays that pH's between 6.28 and 8.10 made no difference in the toxicity of rotenone; they found, however, that toxicity increased as temperature rose. These conflicting reports on the effect of temperature may be due to the fact that rotenone degrades more rapidly in warm water than in cold.

In the lake environment, there are a number of other variables that act to either increase or decrease the effective toxicity of rotenones. Turbidity, soft, mucky bottom areas, weed beds, and organic sediments all appear to decrease the killing power of rotenone. The presence of a thermocline may prevent rotenone from reaching all areas of a lake, thus reducing efficiency. Underwater springs and surface outlets sometimes provide refuge for fish and invertebrates.

APPENDIX C

GLOSSARY

GLOSSARY

ADI	Acceptable Daily Intake of a material which should protect human health. Given as a mg of material per kg of body weight per day.
anoxic	deprived of oxygen
CPUE	Catch per unit effort
cube'	Common name for ground, dried roots (especially of *Lonchocarpus* sp.) containing rotenone.
DDT	an insecticide, dichlorodiphenyltrichloroethane, $(ClC_6H_4)_2CHCCl_3$
derris	Common name for ground, dried roots especially of *Derris elliptica*) containing rotenone.
eutrophic	designating or of a lake, pond, etc. rich in plant nutrient minerals and organisms but often deficient in oxygen in midsummer.
hypolimnion	the lower most, noncirculating layer of cold water in a thermally stratified lake, usually deficient in oxygen.
IP	intraperitoneal
LC50	median lethal concentration; the concentration of a toxin in water that kills 50% of the test animals in the water within a specified time (usually 24, 48, or 96 hours). Usually expressed in ppm.
LD50	median lethal dosage; the dosage of a toxin that when fed or injected kills 50% of the test animals. Usually expressed as mg of toxin per kg of the test animal's body weight.
macrophytes	plant forms, individuals of which can be observed with the unaided eye.
mfo	mixed function oxidase
MSY	maximum sustainable yield
NOEL	No Observable Effect Level for a material exposed to test organisms.

oligotrophic	designating or of a lake, pond, etc. poor in plant nutrient minerals and organisms and rich in oxygen at all depths.
ppm	parts per million, usually by weight. 1 ppm equals 1 mg/l.
RPAR	Rebuttal Presumption Against Registration list for protests against chemicals that the US Environmental Protection Agency has registered and labeled for use.
SNARL	Suggested No Adverse Response Level.

APPENDIX D

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